Archives of Dermatology and Syphilology

VOLUME 48 DECEMBER 1943 NUMBER 6

COPYRIGHT, 1943, BY THE AMERICAN MEDICAL ASSOCIATION

ACTUAL CAUSES OF CERTAIN OCCUPATIONAL DERMATOSES

A STUDY OF 527 CASES, WITH SPECIAL REFERENCE TO DERMATOSES DUE TO MINERAL OILS

JOSEPH V. KLAUDER, M.D.

PHILADELPHIA

A study was made of 1,113 cases of cutaneous diseases in patients observed by me who presented claims for compensation under the Pennsylvania law. My purpose was to determine outstanding causal factors of occupational dermatoses, in order to discuss these factors by groups in relation to a practical outline of teaching occupational dermatoses.[1]

Of the 1,113 patients 909 were males and 204 were females. The diseases of 527 were diagnosed as occupational [2] in origin and those of 586 as nonoccupational. These figures do not include conditions encountered during the inspection of employees at various plants.

The nonoccupational group comprised thirty-nine different diseases of the skin. The most frequent were: eczematous dermatitis, tinea of the hands and feet, pyoderma, pityriasis rosea, miliaria, seborrheic dermatitis, lichen planus, urticaria, herpes zoster and erythema multiforme. Such diseases as syphilis (early and late lesions), pemphigus vulgaris and lupus erythematosus were observed.

Diagnosis of the occupational group was made after a thorough history taking, complete physical examination and consideration of the nature of the work and substances to which the claimant was exposed. Patch tests were performed when indicated. Many workers ascribe to their work all variety of diseases of the skin appearing during employment and designate it by adding the word "poison" or "itch" to the name of the material handled—thus "brass poisoning," "fur itch," etc.

The actual causes of the occupational dermatoses [3] were classified into groups. Some writers, reporting on a large number of dermatoses, have emphasized occupation in relation to cause. Although the two cannot be separated, I believe it is preferable to emphasize actual causes, since attention is focused on these causes, preventive and investigative measures are suggested and teaching of the subject is facilitated.

This paper was written in cooperation with the Council on Industrial Health of the American Medical Association and with the Committee on Industrial Dermatoses of the American Medical Association.

1. Klauder, J. V.: Teaching of Occupational Dermatoses, Arch. Dermat. & Syph. **48**: 153-158 (Aug.) 1943.

2. Some of the dermatoses included in this figure, although occupational, were not compensable under the Pennsylvania law.

3. Patients with diseases in this group represent many trades and occupations comprising workers in a highly industrialized region such as Philadelphia and vicinity.

Actual causes of 504 of the 527 occupational dermatoses are classified in the following groups:

	No. of Cases	Percentage
Trauma and accidental injury	131	24.8
Primary irritants (acids, alkalis, solvents [nonaqueous])	128	24.2
Cleansing agents applied to the skin	86	16.3
Sensitizing substances	66	12.5
Petroleum products	45	8.5
Wet work (water alone, soap and water, and alkaline salt detergents)	48	9.1

In addition, there were 21 cases (3.9 per cent) of occupational folliculitides caused by chlorinated hydrocarbons, vegetable oil and dust.

It was possible to place the causes of all but 2 of the 527 dermatoses in these groups. These 2 were erosio interdigitalis blastomycetica in a baker and a pronounced eruption of herpes simplex which occurred in an employee in a foundry each time he had fever as part of "zinc chill."

All writers emphasize the role of alkalis, lime, cement, metal "pickling" and plating solutions, certain acids, compounds of chromium, dyes and their intermediates and especially solvents (nonaqueous) in causation of occupational dermatoses.

I would stress the role of solvents, especially turpentine. Solvents are extensively used throughout industry.[4] According to Mellan,[5] there is an annual production in the United States of about 1,600,000,000 pounds (726,000 metric tons) and an industrial development of over one hundred different solvents. Some solvents are commonly used in the household, and many, especially turpentine, are used as hand cleansers. Most solvents are primary irritants, and turpentine has a high sensitizing index.[6]

TRAUMA AND ACCIDENTAL INJURY

The two most frequent causes were trauma and accidental injury—131 cases (percentage of incidence, 24.8) and primary irritants—128 cases (percentage of incidence, 24.2).

Diseases of the skin resulting directly or indirectly from trauma and accidental injury are many and varied.[7] They include most infections, dermatoses from

4. The lacquer industry stands first in consumption of solvents, although the new types of coating materials made from synthetic resins are displacing solvents for this purpose.

5. Mellan, I.: Industrial Solvents, New York, Reinhold Publishing Corporation, 1939.

6. It was shown by Burckhardt and Schaaf (Burckhardt, W., and Schaaf, F.: Klinische Erfahrungen und chemische Beobachtungen über die Terpentin-Hautschädigung und ihre Prophylaxe, Ztschr. f. Unfallh. **31**:124-137, 1937) in patch test studies with pinene fractions of turpentine oil that the irritating properties of turpentine are inherent in the pinenes and possibly also in other related products. The pinene components varied depending on the source. The dextrorotatory pinenes (chief component in American turpentine) were more irritating than the levorotatory pinenes (chief component in French turpentine). Slightly toxic pinene fractions could be changed by ultraviolet irradiation to highly colored toxic products. Turpentine when exposed to air becomes yellow, and its toxic properties are increased by autoxidation of the pinene fractions. In Burckhardt and Schaaf's study it was found that not every yellow oil was toxic but every toxic oil was yellow. Z. Pertuz (Arch. f. Dermat. u. Syph. **152**:617, 1926) and others stated the opinion that dermatitis from turpentine always connotes sensitization and always previous exposure. The degree of sensitization, however, varies. A worker reacting to a 50 per cent dilution may tolerate a working contact, whereas one reacting to a 1:100,000 dilution may experience an attack of dermatitis after merely entering a freshly painted room.

7. (*a*) Engelhardt, W.: Die Bedeutung des Traumas für Entstehung und Verlauf von Haut- und Geschlechtskrankheiten und dessen gutachtliche Beurteilung, Arch. f. Dermat. u. Syph. **180**:14-41, 1940. (*b*) Riecke, E.: Trauma in Dermatology, München. med. Wchnschr. **83**:507-510, 1936.

mechanical pressure and friction and many of the so-called occupational stigmas. The sixteen different diseases of the skin arising from trauma and accidental injury in this study are divided into the following subgroups. Many were the natural and direct result of a traumatic injury by accident. Dermatitis resulting from splashing of a caustic solution could have been classified in the primary irritant group. It resulted, however, in the legal sense from an accident. In others, traumatic injury played a contributory role. If such a role is considered, accidental injury becomes an important cause of occupational dermatoses—more important, I believe, than is usually considered.

Pyoderma (41 Cases).—The following lesions, usually on the hands, resulted from injury, frequently minor, from various causes: simple pyogenic lesions of the impetiginized type; granuloma pyogenicum, and infectious eczematoid dermatitis.

Dermatitis (46 Cases).—1. This was caused by an occlusive dressing, discharge or adhesive plaster or was initiated by a wound or burn (24 cases). The dermatitis did not remain localized to the area of onset in all patients. 2. Dermatitis venenata from injudicious treatment of injury or burn or as an allergic reaction to a medicament (trinitrophenol, butacaine [butyn] sulfate) (5 cases). 3. Dermatitis caused by splashing of caustic solution, hot water or oil or by a leaking pipe (10 cases). 4. Chronic eczematous dermatitis (7 cases) resulting from mechanical pressure or friction of a tool, mask or hard object or the article of work. The skin of the affected part was usually dry, scaly, thickened or calloused. One patient, a baker, had dermatitis of the wrists and of two fingers. He used the extended wrists to roll dough and the affected fingers to pry under the layer of dough. Dermatitis of the fingers in another patient was caused by friction on sandpaper.

Ulcer of the Leg (14 Cases).—This resulted from injury, usually severe, in men over 40 years of age, who frequently had varicose veins. Dermatitis usually accompanied the ulcer. The course was protracted and the disability prolonged.

Herpes Zoster Traumaticus (5 Cases).—In 1 case a man bumped the left side of his head against a hard object; pain at the site of injury soon disappeared and he continued to work. Dull headache confined to the left side ensued the same day and became progressively worse until the appearance of herpes zoster. The eruption affected the left side of the head and appeared on the fourth day.

There are a number of reports in the European literature of herpes zoster appearing after injury[8] which may be trivial. Many writers have subscribed to the relationship. Some have stated the opinion that the virus, the alleged cause of the disease, is introduced at the site of injury and results in an ascending infection.

Alopecia Areata (1 Case).—A worker bumped the side of his head against a moving belt; this caused a brush burn, which soon healed. A tingling sensation persisted for four days, at which time alopecia areata appeared at the site of injury.

Psoriasis (1 Case).—The influence of trauma in localizing lesions of psoriasis is well known. In 1 patient the disease appeared at the periphery of a granulating wound. There were lesions of psoriasis elsewhere at the time of injury.

8. (*a*) Ammann, E.: Herpes zoster ophthalmicus traumaticus, Klin. Monatsbl. f. Augenh. **95**:793-796, 1935. (*b*) Fox, H.: Herpes Zoster Following Osteopathic Manipulation of the Spine, J. Cutan. Dis. **34**:773, 1916.

Anthrax (2 Cases).—This disease has long remained an industrial hazard. Smyth [9] studied the geographic distribution, incidence and source of infection of anthrax in the United States from 1919 to 1938. Hides and skins were the cause of anthrax in 38.9 per cent of cases in which the source of infection could be determined. Almost all goat skins tanned in the United States are imported from countries in which anthrax is more or less prevalent. The percentages of other sources were: agriculture, 21.9; wool, 17.9; animal hair, 7.4; shaving brushes, 6.4; transportation (longshoremen, truckers, etc.) 2.4; nonindustrial, 3.1. The studies of Smyth show no decreasing trend in the incidence of anthrax, at least in the Eastern states. The percentage of fatal cases, however, has decreased. This is attributed almost entirely to the occurrence of the disease in organized industries, where early diagnosis and prompt treatment are facilitated.

Anthrax will remain an industrial hazard until a satisfactory method of disinfecting hides and skins has been found. Hides, wool and animal hair are imported under consular certificate that they are from districts free from anthrax. Smyth [10] and others, however, have isolated anthrax spores from such certified material. In England the disease has decreased since the creation of a government-supervised disinfecting station, where all raw wool and undressed hair are disinfected at minimum cost to the importer.

In 1 case in this study the only clinical symptom suggestive of anthrax was an unusual amount of edema surrounding the lesion. This feature and the occupation of the patient, a haircloth weaver, suggested the making of a smear and culture, which confirmed the suspicion of anthrax.

Erysipeloid (7 Cases).—I reported elsewhere [11] 100 cases of erysipeloid (none of which are included in this study), in 88 of which the disease was caused by an occupational injury. Erysipeloid almost always arises at the site of an injury, trivial or otherwise, usually of the hand. The infection, most common in abattoir workers and fish handlers, also occurs in persons handling dead animal matter—fertilizer, glue, bones, etc.

An employee scratched his finger when handling dressed turkeys; erysipeloid resulted at the site of injury. This source of infection is consistent with the occurrence of Erysipelothrix rhusiopathiae infection in turkeys.[12] An employee of a manufacturer of biologic products stuck his finger with the needle used in inoculating a horse with a pure culture of Ery. rhusiopathiae in preparation of immune serum. Erysipeloid appeared at the site of this injury.[13] A butcher cut his finger on a piece of bone. Erysipeloid ensued, with necrosis of bone. Death resulted six months later from Ery. rhusiopathiae septicemia.[14]

9. Smyth, H. F.: A Twenty Year Survey of Anthrax in the United States: Sixth Report of the Committee on Anthrax, Industrial Hygiene Section of the American Public Health Association, Pittsburgh, Pa., Oct. 18, 1939.

10. Smyth, H. F.: The Anthrax Hazard in Pennsylvania Tanneries, Am. J. Hyg. **2**:346-367, 1922.

11. Klauder, J. V.: Erysipeloid as an Occupational Disease, J. A. M. A. **111**:1345-1348 (Oct. 8) 1938.

12. Rosenwald, A. S., and Dickinson, E. M.: Swine Erysipelas in Turkeys, Am. J. Vet. Research **2**:202-213, 1941.

13. There was a repetition of the same accident five months later. At this time the site of injury was immediately treated with a dressing saturated with immune serum. A second erysipeloid infection did not occur, although such infections do occur. This method, I believe, is more likely to prevent erysipeloid than is application of antiseptics. I observed that such applications did not prevent erysipeloid.[11]

14. Ery. rhusiopathiae was recovered from culture of the blood and was demonstrated in endocardial vegetations. This case was separately reported (Klauder, J. V.; Kramer, D. W., and Nicholas, L.: Erysipelothrix Rhusiopathiae Septicemia: Diagnosis and Treatment, J. A. M. A. **122**:938-943 [July 31] 1943).

Sporotrichosis (1 Case).—A gardener burnt his thumb on the door of the hothouse furnace. The primary lesion appeared at the burnt area, and subsequently other lesions of the disease developed on the forearms.

In the first case of sporotrichosis recorded, by Schenck,[15] the disease occurred in a laborer after an occupational injury. It is an important occupational dermatosis of parasitic origin in farmers, gardeners, nursery men and florists. In Foerster's [16] review of reported cases, the average duration before recognition was found to be four months and there was an approximate average period of five months' disability from the time of acquisition of the disease until the time of recovery.

Other dermatoses arising from trauma and accidental injury observed in this study are the following: bite of insect (1 case); keloid (3 cases); pigmentation following a burn (1 case); tattoo from metal filings (1 case); erythema multiforme secondary to cellulitis (1 case); urticaria as part of serum sickness from injection of tetanus antitoxin (1 case); traumatic avulsion of hair (5 cases) occurring in women whose hair was caught in machinery.

PRIMARY IRRITANTS

Of the 128 patients with dermatoses caused by primary irritants, many were exposed to more than one. Consideration of frequency of exposure to one alone and in conjunction with another determined the arrangement of the following groups, which are listed in order of their importance as causal agents.

Solvents.—Kerosene, turpentine and type wash were the most common solvents causing dermatitis. The occupations most frequently represented were: printing (photoengraving and lithography), especially the cleansing of printing presses with type wash (solvents used to clean the type and presses); painting; wood finishing, and furniture polishing. A conspicuous mechanism of the acquisition of dermatitis was the use of solvents to clean mechanical parts or the article of work or the immersion by hand of this material into a solvent without the use of gloves. This was done by machinists, machine operators and a variety of other workers. Dermatitis in a painter caused by wood alcohol is shown in figure 1.

Alkalis.—Exposure of alkalis occurred from the following sources: electroplating; "pickling" of metals; manufacturing of glass; washing of textile fibers; dyeing; engraving (ammonia water); building trades; manufacturing of asbestos compounds and of bone black, and tanning (calcium oxide).

Chromic Acid and Chromates.—The occupations represented which involved exposure to chromic acid and chromates were: printing trades; developing of blue prints; tanning; dyeing of textiles, and washing of dairy cans.

Pigments and Dyes.—Exposure to pigments and dyes occurred in the manufacture of ink and in its uses in the printing industry; in the dyeing industry; in the finishing of leather, and in the manufacture of wallpaper and colored paper.

Paints, Enamels and Varnishes.—Dermatitis from paints, enamels and varnishes occurred in lithographers, photoengravers, painters, furniture finishers and other workers applying these materials to mechanical parts and to a variety of objects, such as rheostats, batteries and hairpins.

Acids.—Exposure to a variety of acids occurred in the following work: lithography, photoengraving, silver plating, tanning and dyeing. Acid used in electric storage batteries provided another frequent source.

15. Schenck, B. R.: On Refractory Subcutaneous Abscesses Caused by Fungus, Possibly Related to the Sporotrichia, Bull. Johns Hopkins Hosp. **9**:286-290, 1898.

16. Foerster, H. R.: Sporotrichosis: An Occupational Dermatosis, J. A. M. A. **87**:1605-1609 (Nov. 13) 1926.

Dermatitis was also observed from the following materials: formaldehyde, used to sterilize combs in a beauty shop; sulfur dioxide, used in refrigeration, and creosote, used in preserving lumber. Punched-out ulcers, similar to those produced by lime and by chromium compounds, were observed from copper sulfate and from sodium or potassium cyanide in electroplaters.

A worker whose hands were exposed to a strong solution of oxalic acid used for cleansing complained of tenderness, soreness and considerable pain (sleep disturbing) of the ends of the fingers. There were no apparent changes in the skin to explain these symptoms.[17]

CLEANSING AGENTS APPLIED TO THE SKIN

In this group there were 86 cases (percentage of incidence, 16.3). Seventy-eight of the patients used a cleansing agent other than soap, and 8 used soap excessively.

Fig. 1.—Dermatitis caused by wood alcohol in a painter who removed paint with this solvent. Tinea (lesions of which are shown on the palms) complicated the dermatitis.

Of the 78 patients, 69 employed a single hand-cleansing agent and 9 used more than one. Volatile solvents were the most frequent cleansing agents used by these patients and included gasoline, type wash, turpentine, paint thinner, kerosene, naphtha, alcohol and toluene. These were used by painters, printers, machinists, wood finishers, furniture polishers and others to remove paint, varnish, lacquer,

17. These symptoms have been described by other writers. I could find no explanation in the medical literature. Dr. Joseph Comero, department of pharmacology, University of Pennsylvania, suggested the following explanation: Oxalic acid, since it readily combines with calcium, may through local action disturb the calcium-potassium ratio of the tissues, resulting in potassium nerve stimulation. This explanation is consistent with the conclusions in Klauder and Brown's study (Klauder, J. V., and Brown, H.: Experimental Studies in Eczema, Arch. Dermat. & Syph. **19**:52 [Jan.] 1929) of the role of the nervous system and of the calcium-potassium ratio in altering cutaneous irritability in the rabbit. In treatment, calcium administered orally and intravenously is suggested. In prevention, in absence of mechanical measures, immersion of the hands in 5 to 10 per cent solution of calcium lactate is indicated.

shellac, glue, cement, ink and dye. Other cleansing agents used were: calcium hypochlorite, by dye house employees and by "seasoners" in tanneries, who also used ammonia water; sodium cyanide, used to remove stains of silver nitrate by men making mirrors and by lithographers; solution of sodium hypochlorite (Javelle water); pumice, and soap powder. Dermatitis caused by turpentine used to remove paint from the hands and forearms is shown in figure 2.

In the 8 patients with dermatitis from excessive use of soap the eruption resulted from washing the hands as much as fifty times daily during the work period. These patients included bakers and others of whom clean hands are required to handle the object of work or to write records.

Included in the group of 128 patients with occupational dermatoses from primary irritants were 23 who used a harmful hand-cleansing agent, usually the primary irritant to which they were exposed in their work. These 23 patients are not included in the group of 86 with dermatitis from cleansing agents applied to the skin.

Fig. 2.—Dermatitis caused by turpentine in a painter who used this solvent as a hand cleanser.

Elsewhere [17a] I discussed the role of some cleansing agents in causation of occupational dermatoses and discussed harmless methods of cleansing the skin.

SUBSTANCES CAUSING SENSITIZATION DERMATITIS

There were 66 cases of sensitization dermatitis (percentage of incidence, 12.5).[18] Sensitization dermatitis from petroleum products (7 cases), anticorrosion oils (6 cases) and linseed oil (1 case) are included in this figure and are separately discussed.

17a. Klauder, J. V.; Gross, E. R., and Brown, H.: Prevention of Industrial Dermatitis, with Reference to Protective Hand Creams, Soap and the Harmful Role of Some Cleansing Agents, Arch. Dermat. & Syph. **41**:331-356 (Feb.) 1940.

18. Cases of dermatitis caused by chromates and by turpentine were placed in the primary irritant group. Routine study was not made in order to determine if dermatitis from these causes was the expression of primary irritant action or of sensitization. If the concept of Perutz [6] is correct, all dermatitis from turpentine should be classified as sensitization dermatitis.

A powder used as a germicidal detergent for glasses caused dermatitis in some restaurant employees washing glasses (10 cases). The ingredient causing sensitization was not determined, although one, N, N-dichloroazodicarbonamidine, was suspected.

Some operators of a knitting machine making nylon hosiery had dermatitis of the exposed skin caused by the solution through which the thread ran (7 cases). The ingredient causing dermatitis was not determined, but pine oil was suspected.

A worker engaged in insulating wires had dermatitis of the hands. Patch tests were performed with the insulating covering of seventeen different wires; two (Holyoke nos. 853 and 855) elicited positive reactions.

A varnish made from the oil extracted from cashew nuts caused dermatitis in 1 patient. This varnish has high insulating and moisture-resisting properties and was used on electric coils.

Four men engaged in mixing a number of ingredients used to coat colored paper and cardboard had at irregular intervals dermatitis of the hands and forearms. Patch tests performed with each ingredient gave negative results, but when the ingredients were mixed together the results were positive.

In manufacturing laminated bakelite (phenol-formaldehyde resin) products, men operating the molding press and others who handle the laminated cloth become sensitized to the resin. These workers are exposed to bakelite dust, which causes a dust eruption, folliculitis (later discussed). Sensitization dermatitis (6 cases), which especially affects the face and eyelids, was less commonly observed than folliculitis.

A clerk used a proprietary ointment to remove stains caused by hectograph paper. He became sensitized to the preparation, and dermatitis of the hands resulted.

A gardener who had a widespread dermatitis was allergic to the leaves of chrysanthemums and also to four varieties (ragweed, wild feverfew, burweed marsh elder and yarrow) of wild vegetation common in Pennsylvania.

Dermatitis was observed from the following sources: vanilla (2 cases), in a vanilla bean sorter and in an employee making beverage; cinnamon (2 cases), in bakers; insecticide (3 cases), in employees engaged in its manufacture; glue (1 case), in a radio assembler; methylaminocresol sulfate (2 cases), in photographers; methyl and ethyl acrylate (2 cases), in employees manufacturing these chemicals; trinitrotoluene (TNT), in an employee in a plant manufacturing explosives; Suprecool permanent wave lotion (1 case) and three different scalp lotions (1 case), in beauty parlor employees, and Rhus (4 cases).

One worker had an outbreak of urticaria after handling burlap bags. Solar erythema attributed to photosensitizing action of pitch was observed in 2 roofers.

DERMATOSES DUE TO MINERAL OILS

In this study there were 45 cases of dermatoses caused by petroleum products (percentage of incidence, 8.5). These dermatoses included oil folliculitis (36 cases), generalized melanosis (1 case), melanosis, keratosis and epithelioma (1 case) and nonsensitization dermatitis from cutting oil (7 cases). Sensitization dermatitis from petroleum products is not included in these figures.

In a discussion of mineral oil dermatoses, it is pertinent to consider types of oils and their admixtures to which workers, particularly in the metal working industry, are exposed.

Metal Working Oils.—Mineral oil for metal working is employed in cutting operations (milling, boring, etc.) in order to remove metal cuttings and to cool the cutting tools. For this purpose, cutting oil is employed, of which there are two types: straight, or insoluble, and soluble. Straight, or insoluble, oil is used as delivered by the manufacturer or, perhaps, diluted with mineral oil. Soluble oil is mixed with water before use.

For metal working in which no cutting is involved both straight and soluble oils are used. For quenching, tempering and annealing operations, in which a liquid bath is necessary, only mineral oil is employed. To protect the finished work from surface corrosion, rust-preventive oils or compounds are used.

Straight, or Insoluble, Oils.—These comprise a mixture of a mineral oil of the paraffinic or naphthenic type with an animal (lard, fish, degras) or a vegetable oil (olive, cottonseed, rape, rosin). To these oils may be added sulfur, chlorine up to 5 per cent and inhibitors (to prevent rancidity), comprising phenols, cresols and nitrobenzene. In sulfur base cutting oils (sulfurized oil), sulfur is heated with fatty oils (animal or vegetable), in order to effect a loose combination of the sulfur with the unsaturated oil, and subsequently mixed with mineral oil, or sulfur is dissolved directly in the mineral oil. Oils of this type as used contain up to 4 per cent by weight of sulfur and are employed in cutting operations of hard metal pieces and harder alloys. Sulfurized oil for the past fifteen years has largely replaced so-called mineral lard oil (mineral oil 90 per cent and lard oil 10 per cent), which, in turn, replaced straight lard oil.

Soluble Oils (Water-Soluble Oil, Cutting Emulsions).—These are mixtures of mineral oil and oil-soluble and water-soluble soaps of fatty or petroleum acids saponified with alkali, to which is added water; the result is a creamy emulsion ("soap" in the parlance of the worker). Volatile matter (alcohol, glycophenol, nitrobenzene, cresol, kerosene or turpentine) up to 10 per cent may be added. Organic acids or alkalis may also serve to inhibit the growth of bacteria. Soluble oils are employed in cutting operations on softer metal and in operations in which carbide tools are used. Since volatile matter in soluble oils is often lost by evaporation, which renders the cutting oil unstable, such matter has been replaced in the oils now in general use by less volatile material, such as alcohols of higher boiling point. Some soluble oils are largely composed of sulfonated oil. Both soluble and sulfurized oils contain vegetable or animal oil, to which antiseptics or deodorizers may or may not be added. Some sulfurized oils contain no fatty oil.

Rust-Preventive Compounds[19] *(Slushing Oils, Rustproof Oils, Anticorrosion Oils).*—These consist of mineral or fatty oils and greases with or without the addition of waxes or soaps. The waxes employed are spermaceti, cetyl palmitate, carnauba and beeswax. Other compounds are: (1) ordinary mineral oils, petrolatum or mixtures of rosin or rosin oil with mineral oil or with emulsions of oil in water; (2) crude hydrous wool fat or mixtures of hydrous wool fat and mineral oil (wool fat 10 per cent and kerosene 90 per cent); (3) heavy fractions of mineral oil containing asphaltic substances, and (4) cylinder oils containing zinc naphthenate and ceresin. Solvents which may be incorporated are: naphtha, trichlorethylene, kerosene, Stoddard solvent (straight run petroleum naphtha with a flash point above 100 F.), benzene and acetone. Antioxidants which may be used are: potassium dichromate (and other salts of chromic acid), phenolic derivatives,

19. The use of rust-preventive compounds has increased incident to greater industrial activity arising from the defense program. There is little information regarding these compounds in medical or in technical literature.

hydroxysulfides, alkyl sulfur-containing compounds, phosphorus compounds, amino compounds and other nitrogen compounds.

A commonly used straight mineral anticorrosion oil is mineral seal oil, a light oil with a distillation range between that of kerosene and that of spindle oil.

Sensitization Dermatitis from Anticorrosion Oils and Petroleum Products.—Sensitization dermatitis was observed from anticorrosion oils, the ingredient which caused the dermatitis not being identified (6 cases); from mineral seal oil contained in an anticorrosion oil (7 cases), and from other products of petroleum later discussed.

In these patients the dermatitis was clinically of the sensitization type, appearing on the hands and forearms and in some on the face, usually months after initial exposure. Patch tests with the offending oils elicited positive reactions in these patients and gave negative results in persons used as controls. Tests with mineral seal oil were performed with samples from two different refineries. The samples were free of admixtures and were obtained from naphthenic and paraffinic types of crude petroleum.

Three of 20 women handling pistons covered with anticorrosion oil became sensitized.[20] It was observed that the hands were less affected than the forearms or face. After disappearance of the dermatitis after brief cessation of work, some became less sensitized [21] and were able to resume the same work without a recurrence or with slight recurrence of the dermatitis.

One worker was sensitized to mineral seal oil which was mixed with lubricating oil and used as an anticorrosion oil on ball bearings. He had dermatitis of the hands and face. He reacted positively to a patch test on the arm with mineral seal oil and negatively to one with the lubricating oil. After recovery, he resumed contact with mineral seal oil but to a less degree. There was a mild recurrence of the dermatitis, consisting of a few small patches on some fingers. A second patch test with samples of mineral seal oil was performed at the same sites as the first. These tests produced positive reactions of the same degree as the first. Apparently the skin of the hands and not that of the arms became less sensitized to mineral seal oil. Since mineral seal oil approaches the distillation range of spindle oil, a patch test was performed with spindle oil. The reaction to this test was negative.

Another worker handling oil-covered ball bearings had dermatitis of the hands and face. Patch tests were performed with samples of petroleum products which were free of admixtures. The results were: with mineral seal oil, 3 plus; with spindle oil (paraffin oil of viscosity 62), 3 plus; with machine oil (motor oil), 3 plus; with heavy liquid petrolatum U. S. P., 1 plus; with naphtha (high solvency) in a 10 per cent dilution in olive oil, negative. Additional evidence of sensitization was a slight flare-up of the dermatitis of the face soon after application of these tests.

To my knowledge, dermatitis from mineral seal oil has not been reported in medical literature. Schwartz [22] in his study of dermatitis among employees in oil

20. In 1 woman dermatitis first appeared around the ears; this was attributed to contact with the hands when she arranged her hair while working. One phase in treatment consisted of the wearing of a hairnet and refraining from touching the face. She resumed work; the only recurrence was circumscribed lesions of mild dermatitis of the hands.

21. This illustrates what workers call becoming "hardened" to the work. Gougerot and Basset (Bull. Soc. franç. de dermat. et syph. **46**:1329, 1939) reported instances of workers becoming desensitized to vanilla. Schwartz observed instances of desensitization of industrial workers to a variety of cutaneous allergens.

22. Schwartz, L.: Dermatitis in Oil Refining, in Schwartz, L., and Tulipan, L.: Skin Hazards in American Industry, Bulletin 215, United States Treasury Department, Public Health Service, 1934, pp. 11-17.

refineries stated that occasionally an employee becomes sensitized to petroleum distillates. He did not mention the nature of the distillates.

Zinc chromate in a rustproofing compound that had been applied to metal parts was the cause of dermatitis of the hands, forearms and face in a worker handling these parts.

Dermatitis from Cutting Oil.—Dermatitis from cutting oil is not common. Only 1 man had dermatitis of the hands in a survey I made of 128 men exposed to insoluble (sulfurized type) cutting oil, and in 4 the hands and forearms were unusually dry. Cutting oil was attributed as the cause of nonsensitization dermatitis in 7 of the 527 cases of occupational dermatoses now reported. In some, judgment of the role of oil was difficult in view either of an associated tinea of the hands or of methods employed in cleansing the hands.

Eczematogenous action of cutting oil in addition to sensitization to petroleum products may be attributed to the following factors: allergic reaction to admixtures (inhibitors, antiseptics, deodorizers and antioxidants); rancidity of animal or vegetable oils; prolonged contact with water in soluble cutting oils and to a less degree with the alkali content of such oils [23]; secondary eczematization of lesions of oil folliculitis. I observed such lesions becoming confluent, forming an eczematous patch. Considering the dilution of soluble cutting oil with water, it is doubtful if any ingredient acts as a primary cutaneous irritant.

Sulfur contained in sulfurized oils, now in considerable use, is not sufficient to act as a cutaneous irritant nor is it allergenic. The p_H values of these oils indicate that they are neutral or slightly acid.

Wedroff and Dolgoff [24] concluded from their study that there is no parallelism between the eczematogenous action of mineral oil and its disposition to cause folliculitis. From their observation of workers exposed to mineral oil of the naphthenic type, the incidence of eczematous dermatitis was no greater than its incidence in general. Among such workers, dermatitis was observed only in those contacting oil with an admixture of irritating substances.

In Hopf's [25] observation, dermatitis from mineral oil was not common except in eczematic persons. In contrast to studies herein reported, he was not able to demonstrate an allergenic property of mineral oil.

Solar Dermatitis.—Mineral oil may cause dermatitis by sensitization to light. Solar dermatitis from this source was not observed in this study but was observed in 2 patients; both were roofers who handled pitch.

Melanosis, Keratosis and Epithelioma.—The mineral oils of North America are low in carcinogenic property.

Melanosis, keratosis and epithelioma, constituting the "shagreen" skin, especially described by White,[26] occurring in "mule spinners" in England exposed to shale oil is rarely seen in industrial workers in the United States exposed to

23. The p_H of all insoluble cutting oils, both with and without sulfur, that I determined was on the acid side. The p_H of a soluble cutting oil—10 parts of a mixture of petroleum sulfonate and mineral oil in 70 parts of water was 9.6. The eczematogenous action of soluble cutting oils, so far as concerns prolonged contact of the skin with alkali, is perhaps comparable to such action of soap and water.

24. Wedroff, N. S., and Dolgoff, A. P.: Zur Frage der Pathogenese der Oelacne, Arch. f. Gewerbepath. u. Gewerbehyg. **6**:428-436, 1935.

25. Hopf, G.: Ueber gewerbliche Oelschädigungen der Haut, Aerztl. Sachverst.-Ztg. **43**: 229-241, 1937.

26. White, R. P.: The Dermatergoses, or Occupational Affections of the Skin, ed. 4, London, H. K. Lewis & Co., Ltd., 1934, p. 208.

lubricating oils.[27] This syndrome, as shown in figure 3, was observed in 1 patient. He was a man aged 64 who had worked in an oil refinery as a paraffin wax "presser" for twenty-four years. He first noticed "warts" on his arms three years prior to the time the photograph was made.

Schwartz [28] observed that about 10 per cent of all men working with oils and greases have small flat brownish papillomas on the skin exposed to oil or grease. He attributed these lesions to the keratogenic action of some mineral oils.

In White's [26] observations, "flat, white warts" which never became malignant are common in shale oil workers. Melanosis in workers exposed to mineral oil is attributed either to the direct pigmentary action of the oil or to the presence of photosensitizing substances. Generalized melanosis was observed in 1 man who also had diffuse lesions of oil folliculitis. This patient was exposed to cutting oil only for two months.

In the survey I made of men exposed to cutting oil, 5 per cent had keratotic lesions on the hands and forearms. For 2, the duration of exposure to oil was six and fourteen months respectively; for those remaining, exposure varied from ten

Fig. 3.—Melanosis, keratosis and squamous cell epithelioma in a paraffin wax presser. He did this work in an oil refinery for twenty-four years.

to twenty-four years. The ages of the men with keratosis varied from 35 to 50 years. Keratosis was present with and without oil folliculitis and in the absence of melanosis.

It is difficult to separate the keratogenic properties of mineral oil from its carcinogenic properties. It is therefore desirable in prevention of epithelioma to regard keratosis and possibly, too, melanoma as evidencing carcinogenic action of mineral oil.[29]

---

27. According to Wedroff and Dolgoff [24] this syndrome is unknown in the Soviet Union. They attributed this to the fact that no products of coal or slate distillation are mixed with naphthenic oil. Hopf [25] reported the syndrome as rare in Germany. He observed it in 3 workers exposed to oil mixtures containing various hydrocarbons. Most writers express the opinion that the syndrome results from exposure to inadequately purified oils.

28. Schwartz, L.: Dermatitis from Cutting Oils, Pub. Health Rep. **56**:1947-1953, 1941.

29. Such lesions may be prodromal to the development of epithelioma. It is advisable therefore that workers with keratosis or melanoma apply to the exposed skin equal parts of olive oil and wool fat (anhydrous) as protection from the carcinogenic action of mineral oils. This is in accordance with the results of experimental studies of the Tworts as discussed in a previous communication.[17]

Pyogenic Lesions.[30]—The hands of workers employed in metal-cutting operations are likely to be cut with flying chips, and infection may develop. Such lesions were observed in this study and are included in the trauma and accidental injury group.

Rosenberger [31] and others have shown that mineral oils are not suitable mediums for the growth of micro-organisms. The importance of adding an antiseptic to lubricating oils not containing an animal or vegetable oil I believe has been exaggerated. The property of mineral oil causing oil folliculitis is not inherent in its bacterial content.

Oil Folliculitis (Oil Acne, Oil Boils).—Oil folliculitis is the most important and most frequent dermatosis in workers exposed to lubricating oils and greases. It has been outstanding among occupational dermatoses since its first description by Husemann,[32] in 1860. Little has been added to knowledge regarding it since Leloir's [33] comprehensive report in 1889.[34]

The syndrome of oil folliculitis comprising comedos, follicular horny papules, pustules, pyodermas and acne-like papules, appropriately called by the French *bouton d'huile,* is well known and need not be detailed (figs. 4 and 5).

The 36 patients with oil folliculitis whose cases are included in the 527 cases of occupational dermatoses were claimants under the Pennsylvania law. The oil folliculitis was severe, in some necessitating layoff from work. Most of these patients were white men; a few were women. The disease is said not to occur in Negroes.[35]

To determine the incidence of oil folliculitis among workers exposed to lubricating oil, I examined 128 men operating automatic screw machines who were exposed to sulfurized oil. The degree, represented by grade, of follicultis with the percentage of incidence that these men presented was as follows:

Grade 1: only comedos, variable in number, on the hands and forearms. The incidence of this grade was 45.3 per cent (fig. 4).

Grade 2: comedos and not more than eight papules or pustules. The incidence of this grade was 22.6 per cent.

Grade 3: comedos and more than eight papules or pustules; severe pyoderma, or scars resulting from destruction of the follicular orifices. The incidence of this grade was 13.2 per cent (fig. 5).

Normal: none of the aforementioned lesions of oil folliculitis. This comprised an incidence of 18.7 per cent.[36]

There were 4 men with sufficiently severe oil folliculitis of grade 3 to require layoff from work for proper treatment. This would suggest an incidence of incapacitating oil folliculitis of about 3 per cent.

30. Pyodermas complicating oil folliculitis are not included here.

31. Rosenberger, R. C.: Bacteriologic Study of Cutting Oils Causing Skin Lesions, New York M. J. **116**:337-379, 1922.

32. Husemann, cited by Lewin, L.: Ueber allgemeine- und Hautvergiftung durch Petroleum, Virchows Arch. f. path. Anat. **112**:35-69, 1888.

33. Leloir, H.: De la folliculiti et perifolliculiti des fileurs et rattacheurs (bouton d'huile), Ann. de dermat. et syph. **10**:671-678, 1889.

34. Leloir accurately described the lesions of the disease, its incidence, its evolution and its histologic features. He pertinently wrote that the best treatment was its prevention and emphasized the importance of thorough washing and use of clean clothes. He urged the municipal authorities of Lille to provide public baths for workers in the spinning mills. His paper constitutes an interesting aspect of public health and preventive medicine.

35. In my observations, Negroes are not employed in work entailing exposure to cutting oils, in which work the incidence of folliculitis is greater.

36. It is interesting to compare these figures with the incidence given by Leloir,[33] who wrote in 1889 that 5 to 8 of every 10 men engaged as spinners had lesions of folliculitis.

Of the men with folliculitis of grade 3, duration of exposure to oil was usually at least one year. Other than this, no correlation could be made between duration of exposure and degree of oil folliculitis. Some men with folliculitis of grade 2 were

Fig. 4.—Mineral oil folliculitis (grade 1), showing comedos and small follicular horny papules, the first lesions to appear.

Fig. 5.—Mineral oil folliculitis (grade 3), showing comedos, prominent follicular orifices, acne-like papules and pustules.

exposed only a few days; others, many years. Of the men with folliculitis of grade 1 and of the normal group, some were exposed as long as twenty and twenty-

five years. I believe this suggests a predisposition to oil folliculitis, since it is doubtful if hygienic measures alone would explain this discrepancy.

Mention has already been made of the cutaneous lesions other than oil folliculitis observed in this survey.

Of metal-cutting operations, use of the automatic screw machine entails more than usual exposure of the operator to cutting oil, since the hands and forearms are saturated throughout the work period. The following surveys of the incidence of oil folliculitis include operators of other metal-cutting machines in which there is less exposure to cutting oil and also workers exposed to petroleum products other than cutting oil.

A group of 33 women were examined. Some operated milling machines; others, drill presses. The duration of employment ranged from a few weeks to six months. The incidence was as follows: grade 1, 24.2 per cent; grade 2, 9 per cent; grade 3 (1 woman), 3 per cent. The normal group included 63.6 per cent of the women.

The incidence in 28 men operating a turret lathe was: grade 1, 42.8 per cent; grade 2, 28.5 per cent; grade 3, nil. The normal group was 28.5 per cent.

Oil refinery employees, on account of closed processes, are exposed but little to petroleum products. This does not apply to paraffin wax "pressers" and to some employees in the laboratory. In one refinery, 31 men in the sample room of the laboratory were examined. The hands and forearms of these men are soiled with naphtha and with crude, lubricating and fuel oils. These men are less exposed to mineral oil than are operators of a metal-cutting machine. Of the 31 men, 2 presented grade 1 oil folliculitis and 2 grade 2. The hands and forearms of the remaining men were normal. In Schwartz's [37] study of 81 men (in three refineries) employed as paraffin wax "pressers," 19 had oil folliculitis and 25 had keratosis on the hands and forearms.

Workers exposed to liquid petrolatum ("mineral white oil") were examined. The only place of such exposure available was a candy factory. Hot liquid candy is poured into pans, prior to which the pans are greased by hand with a cloth saturated with refined mineral liquid petrolatum ("technical mineral white oil"). I was informed that the men greasing the pans had "oil folliculitis." Of the 6 men engaged in this work, 2 presented a sparse number of follicular papules on the forearms. Most of the follicular orifices were prominent and had a halo of redness. There were no comedos; the appearance was not that of an acneform eruption from lubricating oil.

Pathogensis: Pathogensis of oil folliculitis [38] concerns physical and chemical action on the follicle.

Some writers have expressed the opinion that the follicle is obstructed by precipitation of paraffin wax, with resulting irritation of the epithelial lining of the

37. Schwartz,[22] p. 14.

38. There is confusion in terminology of petroleum products in medical literature on oil folliculitis. In citing foreign literature I have added the word "wax" after paraffin when the meaning is solid paraffin or paraffin wax. "The term 'paraffin' is often confusing when used in the literature. In some cases it is meant to designate kerosene; in others, paraffin wax; in others, paraffinic hydrocarbons in general, and in still others, medicinal mineral white oil (paraffinum liquidum) [liquid petrolatum U. S. P.]. To avoid this confusion the technical literature of the petroleum industry usually refers to solid paraffins as 'paraffin wax,' to kerosene as 'kerosene' or 'water white refined oil' and to medicinal oil [liquid petrolatum U. S. P.] as 'mineral white oil.' The term 'paraffin' is used to refer to paraffinic hydrocarbons" (Brill, F. A., Atlantic Refining Co.: Personal communication to the author).

walls of the follicles. Arning [39] and also Oppenheim [40] attributed this to the crystaloid nature of paraffin wax. Papel [41] concluded from his study that since pure paraffin wax and refined oil will not produce the disease the chemical action concerned is chiefly due to naphthenic acid and resins present in the mixtures he employed. He stated the belief that oil itself is inactive but that it permits the paraffin wax and its admixtures to enter the follicle. Other investigators attribute the disease to resins and asphalts; still others, to unsaturated combinations. It is to be recalled that oil may contain hydrocarbons of the paraffin series, olefine series, diolefine series, naphthene series and aromatic series; sulfur, nitrogen and oxygen combinations, and resins, asphalts and other chemically unidentified combinations.

In a study of oil folliculitis, Wedroff and Dolgoff [24] applied compresses of mineral oil (naphthenic type) comprising spindle oil with and without the addition of 3 per cent sulfur and medicinal liquid petrolatum. The compresses were applied to the forearms of different persons for periods varying from three to thirty-five days. With spindle oil, folliculitis was produced in 6 of 9 persons in an average of sixteen and a half days; with sulfurized spindle oil in 4 of 6 persons in an average of seventeen days; with medicinal liquid petrolatum in 10 of 20 persons in an average of seventeen and a half days. Folliculitis was produced with unused oil as well as with sterile oil.

From this study, it was concluded that combinations of mineral oil of the naphthenic type are capable of producing folliculitis and that purification of oil cannot render it harmless.

Other writers, notably Hopf,[25] have emphasized physical action inherent to plugging of the follicle by dirt and metal chips, which causes increased production of sebum and horny matter, retention of secretion and secondary infection. Hopf stated the opinion that in this manner chronic oil folliculoses, explicable in the sense of a genuine acne, may develop, and that it is not necessary to assume the effect of a chemical action inherent in a component of the oil. In support of this thesis, Hopf stated that oil folliculitis occurs only rarely among workers in oil refineries but predominates in persons employed in metal-working industries. According to him, chemical action of oil in the pathogenesis of oil folliculitis is inherent in the heavy oil fractions not used in cutting oils.

He was able to produce oil folliculitis experimentally (in conflict with the results of Wedroff and Dolgoff) only with the heavy oil fractions but did not succeed with the more volatile mineral oils used in current cutting oils even when the possibly incriminating components were increased—paraffinic, aromatic and naphthenic hydrocarbons. He believed, in contradistinction to others, that the paraffin component is irrelevant and that the more volatile oil fractions are of little import. This, he stated, is consistent with the observation that the numerous cosmetic ointments containing as a base volatile water-clear liquid petrolatum cause no cutaneous reaction.

Comment: It is apparent that opinions on the pathogenesis of oil folliculitis are conflicting. In discussion the following considerations are pertinent: An acneform eruption that characterizes mineral oil folliculitis is also caused by other substances; it is reported to have been experimentally produced with unused oil, sterile oil and medicinal liquid petrolatum; it was observed in this study from

39. Arning, cited by Ullman, K.: Rohol, Paraffin und Ch-Gruppe des Kohlenteers, in Ullman, K.; Oppenheim, M., and Rille, J. H.: Die Schädigungen der Haut durch Beruf und gewerbliche Arbeit, Leipzig, Leopold Voss, 1926, vol. 2, p. 275.

40. Oppenheim, M., cited by Ullman, K.; Oppenheim, M., and Rille, J. H.: Die Schädigungen der Haut durch Beruf und gewerbliche Arbeit, Leipzig, Leopold Voss, 1926, vol. 2, p. 216.

41. Papel, cited by Wedroff and Dolgoff.[24]

unused, clean oil (in oil refinery employees), although in lessened incidence. Is its greater incidence in workers exposed to cutting oil to be attributed to the physical effects of metal chips and other extraneous matter or to greater and more continuous exposure to the oil itself? I believe that physical action [42] and the seborrheic state (acne vulgaris) play minor roles, excessive hair on the forearms a greater role. From the evidence, I regard chemical action of the oil, predisposition and degree of exposure as the more important causes.

Prevention: Many writers since Leloir have stressed the importance of cleanliness and of minimizing exposure to oil by mechanical measures. These measures are well known and need not be discussed. Mention may be made, however, of the desirability of dispensing clean cloths to workers to be used to wipe oil from the hands and forearms.[43]

After the worker has washed with soap and water, Hopf [25] advised the use of 2 to 4 per cent sodium bicarbonate or dibasic potassium phosphate as an additional agent for degreasing the skin. This is apparently advised for the purpose of saponifying fatty oils. In this event, their use is more appropriate after exposure to soluble oil than to insoluble oil.

Hopf [25] has expressed the opinion that protective ointments containing a wax base afford a certain amount of protection. Schwartz [44] advised use of an ointment containing a vegetable and an animal oil, such as the following preparation (Schwartz's type formula):

	Gm. or Cc.
Anhydrous wool fat	70.0
Castor oil	30.0

Perfume as desired.

Melt the wool fat and mix in the castor oil. Perfume when cool.

I employ two protective applications [45]—one over the other. The first is smeared thickly on the skin of the entire forearm as a base for the second.

	Gm. or Cc.
Zinc oxide	25.0
Kaolin	25.0
White petrolatum	50.0

The second solution is then applied by means of a small brush. It dries and leaves a varnish-like film not easily rubbed off, which repels oil and water.

	Gm. or Cc.
Ethyl cellulose	5.0
Mastic	8.0
Castor oil	1.0
Acetone (technical)	86.0

42. My opinion was asked if in an industrial establishment additional installation of machines to filter cutting oil would cause lessened incidence of oil folliculitis. For reasons discussed, I was doubtful if this result would be achieved. Expenditure of money required for such installation for other preventive measures would more likely reduce incidence of oil folliculitis.

43. One woman had oil folliculitis on the lower surfaces of the thighs corresponding to the bottom of her apron, which dripped oil so that her trousers became saturated. The hands and forearms were free. She explained this to constant wiping with a cloth. Perhaps, too, the hands and forearm were more thoroughly and frequently washed than the thighs.

44. Schwartz, L.: Protective Ointments and Industrial Cleansers, M. Clin. North America 2:1195-1212, 1942.

45. These formulas were published elsewhere,[17] the paste as formula 9 and the solution as formula 7. The use of the two together enhances the protective value of each and obviates the difficulty of removing the ethyl cellulose solution from the skin. Formula 8—polyvinyl acetal resin (Monsanto Chemical Co.) 5 to 10 per cent, castor oil 1 per cent and acetone (technical) 38 to 94 per cent—can also be used for the second application.

After several hours the filmed surface may crack around the wrist and elbow. The two preparations can be reapplied to these areas. To remove both applications, the filmed surface is broken with a cloth or a tissue towel; the paste is wiped off the skin prior to washing with soap and water.[46] These applications are particularly useful in treatment of severe oil folliculitis, since they permit the patient to continue work. In such cases, ichthammol can be added to the paste for therapeutic purposes.

Treatment: Pus if present should be evacuated. Use of compresses is desirable, preferably ones containing an antiseptic, such as solution of boric acid to which is added mecurophen (sodium hydroxymercuryorthonitrophenolate) or solution of aluminum acetate. The use of fatty ointments should be avoided. A shake lotion containing 10 to 20 per cent ichthammol is of value. Ichthammol can also be applied in alcohol. Hopf [25] recommended the application of pure ichthammol to large furuncular lesions. I regard roentgen therapy as helpful.

FOLLICULITIS OF OCCUPATIONAL ORIGIN

This group comprises 21 cases (percentage of incidence, 3.9) of folliculitis caused by chlorinated hydrocarbons (5 cases), linseed oil (1 case), dust (14 cases) and glass wool (1 case).

In addition to mineral oil, folliculitis of the acneform type is caused by coal tar, pitch, asphalt, creosote and chlorinated hydrocarbons. Vegetable oils cause folliculitis not of the acneform type [47]; its occurrence from this source is less common than from mineral oil. Fewer workers are exposed to vegetable oils than to mineral oil. Folliculitis has been reported from linseed oil, rape seed oil [25] and oil of citronella [48] and by Nakauchi [49] from soy bean oil. The soy bean oil mixture causing folliculitis, however, contained mineral oil. Glaser [50] reviewed a rather small number of reports of dermatitis and folliculitis from other vegetable oils and from some animal oils.

Folliculitis from Chlorinated Hydrocarbons.—Folliculitis from chlorinated hydrocarbons was observed in workers (5 cases) making radio condensors and handling wire covered with Halowax, a chlorinated naphthalene, and in another group employed in wiring large switchboards.

The eruption caused by chlorinated hydrocarbons has been described by others [51] and need not be detailed. It is similar to the acneform folliculosis from mineral oil except that the lesions are more generalized and the face more likely to be affected; the papules are larger and more shotlike or cystlike (fig. 6), and suppuration is less.

---

46. Some workers, objecting to the time required (less than five minutes) to apply the two applications, have omitted the second one. The paste, when used alone, more easily rubs off; its surface is somewhat sticky and soils objects in contact with it. This, however, is no objection in operating a metal cutting machine. Others have objected to the sense of stiffness caused by the filmed surface, although movement of the arms is not restricted.

47. Other occupational folliculitides of nonacneform type embrace the follicular papules seen in copra dermatitis and in eruptions caused by nonburrowing mites.

48. Lane, C. G.: Dermatitis Caused by Oil of Citronella, Arch. Dermat. & Syph. **5**:589-590 (May) 1922.

49. Nakauchi, Y.: Ueber die durch industrielle und Oele verursachten, akneähnlichen Hauterkrankungen: durch sog. Shôyu-Abura (Sojaöl) verursachte Follikelerkrankung, Jap. J. Dermat. & Urol. **48**:1, 1940.

50. Glaser, E.: Animalische und vegetabilische Fette und Oele in Bezug auf gewerbliche Hautschädigungen, in Ullman, K.; Oppenheim, M., and Rille, J. H.: Die Schädigungen der Haut durch Beruf und gewerbliche Arbeit, Leipzig, Leopold Voss, 1926, vol. 2, p. 216.

51. (*a*) Jones, J. W., and Alden, H. S.: An Acneform Dermatergosis, Arch. Dermat. & Syph. **33**:1022-1034 (June) 1936. (*b*) Jones, A. T.: Etiology of Acne with Special Reference to Acne of Occupational Origin, J. Indust. Hyg. & Toxicol. **23**:290-312, 1941.

The mechanism in the production of the eruption is not known. The action is probably local rather than produced through absorption, as was originally thought.

Linseed Oil Dermatoses.—The literature on linseed oil dermatoses is scanty. Vokoun,[52] Barnes[53] and Schwartz[54] described the eruption occurring among workers in linseed oil mills as an eczematous dermatitis.

In this study there were 3 Negro workers in the pressroom of a linseed oil mill who had dermatoses. Two presented a diffuse eczematous dermatitis especially affecting the arms, thighs and abdomen. The eruption improved each time there

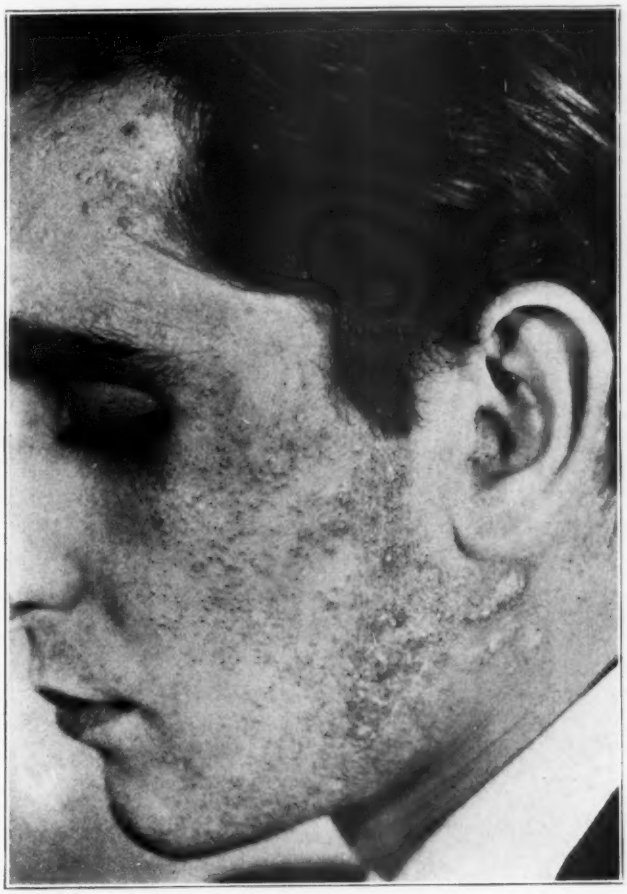


Fig. 6.—Folliculitis (chloracne) from chlorinated naphthalenes, a synthetic wax used to insulate wire.

was a layoff from work and became aggravated when the subject resumed work. Patch tests were performed with the following substances: filtered and unfiltered raw linseed oil, whole flaxseed, ground flaxseed, flaxseed screenings and ground linseed meal. These tests elicited negative responses except for a 2 plus erythematous reaction to linseed meal. Control reactions were negative. The third

52. Vokoun, F. J.: Linseed Oil Dermatitis, Ohio State M. J. **23**:229-230, 1927.
53. Barnes, M. H.: Linseed Dermatitis, J. Indust. Hyg. **13**:49-55, 1931.
54. Schwartz, L.: Dermatitis in the Manufacture of Linseed Oil, in Schwartz, L., and Tulipan, L.: Skin Hazards in American Industry, Bulletin 215, United States Treasury Department, Public Health Service, 1934, pp. 41-45.

patient presented a distinctive eruption composed of discrete, closely aggregated papules that centered around a follicle, as shown in figure 7. They were flat topped, somewhat resembling lichen planus, and diffusely involved the trunk, arms and thighs. At places the papules were confluent, forming patches which were excoriated and eczematized. Patch tests with the aforementioned material gave negative results, except that filtered and unfiltered raw linseed oil reproduced the papular lesions.

A survey was made of the mill employing these men. All the employees, a total of 83 men, of whom 74 were Negroes were examined. In the pressroom the temperature is elevated; the men work wearing only trunks; they perspire freely,

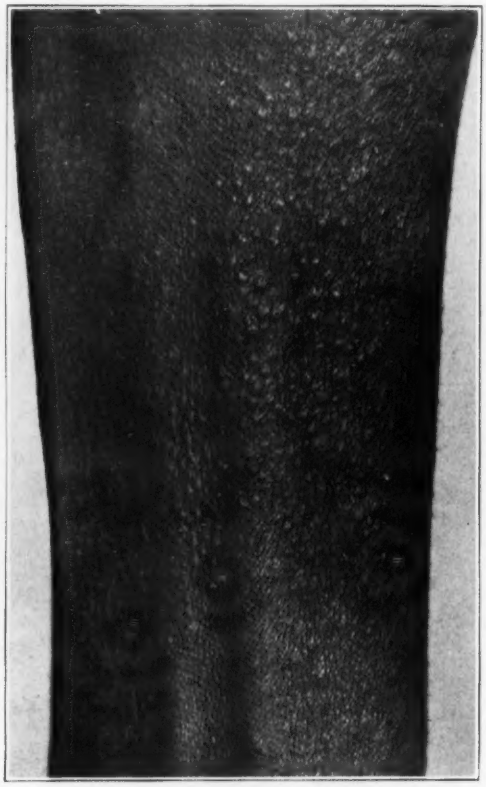


Fig. 7.—Linseed oil folliculitis. Follicular papules on the forearms of a worker in the pressroom of a linseed mill.

and their skin is saturated with linseed oil and meal. Burlap bags are used to remove the oil and meal (this has been suggested as a cause of dermatitis). Two men (Negroes) in the pressroom had a follicular eruption similar (but less pronounced) to that of the patient whose photograph is shown in figure 8.

This study suggests that there are two types of linseed dermatoses [55]: One is linseed oil folliculitis (to which little attention has been called); the other, a dermatitis, an expression of allergic hypersensitivity to linseed meal rather than a reaction

55. This distinction is important from the standpoint of prevention and judgment as to whether the worker should resume the same occupation. Linseed oil folliculitis should be more amenable to prevention by thorough washing at the end of the work day.

to a primary irritant. An allergic basis of the dermatitis is consistent with observations [56] that flaxseed and its derivatives cause other allergic reactions.

Folliculitis from Dust (Dust Eruption).—Dust, comprising a variety of materials, causes plugging of the follicles, which impedes the flow of sebum and sweat and results in ordinary folliculitis and perifolliculitis. Areas of predilection are places where dust is likely to settle—the exposed parts of the skin and moist regions, such as the cubital spaces, the axillary folds and creases of the neck. Under the clothing, favored regions are the lower part of the abdomen and back, around the umbilicus, between the thighs, on the calves and between the toes.

Folliculitis was observed from the following dusts: flour (1 case)—this patient sprinkled flour on slabs of candy and, in addition to folliculitis, he had asthma as an allergic reaction to flour—sand (2 cases); bakelite (9 cases), and sugar (2 cases). Sugar dust is prevalent in sugar refineries and covers the exposed skin and clothing of the workers. Folliculitis in 1 such employee is shown in figure 8.

Fig. 8.—Sugar folliculitis in a worker in a sugar refinery.

Folliculitis associated with eczematous papules affecting the forearms was observed in a worker handling glass wool in the manufacturing of electric refrigerators.

WET WORK

Wet work caused 48 cases of dermatitis in persons in the following subgroups:

(*a*) Workers whose hands or whose hands and forearms were wet with water throughout the work day. There were 22 of these, including workers in dye houses, tanneries, breweries and abattoirs, persons engaged in manufacturing hats, producing textiles, waterproofing canvas and washing rags used for fabric in the manufacture of rubber compounds, and bar tenders, oyster openers, workers in city dairies, sandwich makers and cigar makers. In making cigars, women apply an emulsion of tragacanth in water to the wrapper by means of the fingers. The skin

56. (*a*) Grant, L. R.: Linseed: Report of Six Cases of Flaxseed Sensitization with Review of Literature, J. Allergy **3**:469-477, 1932. (*b*) Jimenez-Diaz, C., and Sanchez Cuenca, B.: Asthma Produced by Susceptibility to Unusual Allergens, ibid. **6**:397-403, 1935.

of the fingers used in this work becomes white, puckered and macerated, evidencing prolonged contact with water.

(*b*) Nurses, barbers, and beauty parlor employees (9 cases). Nonsensitization dermatitis of the hands of these workers was attributed to the wet nature of their work and exposure to soap.

(*c*) This group included 17 workers exposed to soap and water, soap powder and other cleansing compounds containing alkaline salt detergents. These were porters, janitors, scrubwomen, kitchen workers, soda fountain clerks and other workers.

For some of these 17 workers I performed patch tests with all commonly used alkaline salt detergents. The results were negative. Schwartz [57] observed patients who were allergic to trisodium phosphate, and Blaisdell,[58] to silicates of sodium. I believe dermatitis from prolonged exposure to alkaline salt detergents can usually be attributed to impaired function of the skin to neutralize alkali—an "exhaustion" dermatitis. In prevention, therefore, consideration should be given to immersion of the hands at the end of the work day in a weak acid solution, such as 1 to 3 per cent acetic acid, as a buffer application to expedite return of the p_H of the skin to normal. I have discussed this elsewhere.[17]

Downing [59] pertinently wrote: "No worker should be compelled or permitted to keep his hands wet with any solution for hours at a time."

All workers engaged in wet work should use an emollient at the end of the work day.

1934 Spruce Street.

57. Schwartz, L.: Dermatitis in the Manufacture and Use of Alkalis, in Skin Hazards in American Industry, Bulletin 229, United States Treasury Department, Public Health Service, 1936, pp. 69-72.

58. Blaisdell, J. H.: Medicolegal Complications in Dermatology Caused by Massachusetts Workmen's Compensation Act, Arch. Dermat. & Syph. **39**:69-88 (Jan.) 1939.

59. Downing, J. C.: Cutaneous Eruptions Among Industrial Workers: A Review of Two Thousand Claims for Compensation, Arch. Dermat. & Syph. **39**:12-32 (Jan.) 1939.

EFFECT OF ANDROGEN ON THE SEBACEOUS GLANDS OF HUMAN SKIN

HUGO R. RONY, M.D.
AND
SAMUEL J. ZAKON, M.D.
CHICAGO

Physiologic changes in certain areas of the skin at puberty and clinical observations on the skin in cases of eunuchoidism, castration, masculinizing tumors of the ovary and pubertas praecox suggested long ago that sex hormones may in some way be involved in the control of the growth of hair and of the texture of the skin. More recently, as crystalline estrogenic and androgenic substances became available for experimental as well as therapeutic use, efforts were made to disclose effects of these substances on the skin more exactly and in more detail. Thus, valuable information was obtained concerning general effects of estrogenic and androgenic substances on growth of hair in the rat, dog and guinea pig [1] as well as in man,[2] and on the blood vessels of the skin in man.[3]

Since the skin is a complex organ and growth of hair a highly complicated process, the next step obviously is to find out in what way the various specific histologic structures of the skin, and especially of the hair organ, are affected by androgens and estrogens. In this endeavor the response of the human skin is of special importance, in view of the fact that the growth of body hair in man has certain distinctive features as compared with that in experimental animals. The observations described here were made in an attempt to take these points into consideration.

EXPERIMENTAL METHOD

Six boys aged 8 to 9 years were selected for this study. They were all healthy and normally developed except for moderate obesity (4 subjects) and unilateral cryptorchidism (2 subjects). A small portion of the skin was obtained with use of a biopsy punch from the pubic region about 1 inch (2.5 cm.) to the right of the midline. Starting the same day intramuscular injections of 25 mg. of testosterone propionate in 1 cc. of peanut oil were given three times weekly for two weeks, a total of 150 mg. Two or three days after the last injection another specimen of the skin from a site about 1 inch (2.5 cm.) to the left of the midline was obtained in the same manner.

RESULTS

No visible hair growth or other changes were observed in the pubic region or elsewhere after the series of injections. However, comparison of the original sections with those obtained after the administration of the stated amounts of testosterone showed a decided increase in the number as well as in the size of the sebaceous glands in all subjects (figs. 1 to 4). No consistent differences were

From the Department of Endocrinology, service of Dr. J. Isaacs, and the Department of Dermatology, service of Dr. S. J. Zakon, Mount Sinai Hospital.

1. Zondek, B.: Inhibitory Effect of Follicular Hormone on Anterior Lobe of Pituitary Gland, Lancet **1**:10 (April 4) 1936. Gardner, W. U., and DeVita, J.: Inhibition of Hair Growth in Dogs Receiving Estrogen, Yale J. Biol. & Med. **13**:213 (Dec.) 1940. von Wattenwyl, H.: Die Wirkung subkutan implantierter Oestradioltabletten auf das Haarkleid des Meerschweinchens, Schweiz. med. Wchnschr. **71**:1331 (Oct. 25) 1941.

2. Hamilton, J. B.: (*a*) Treatment of Sexual Underdevelopment with Synthetic Male Hormone Substance, Endocrinology **21**:649 (Sept.) 1937; (*b*) Male Hormone Substance: Prime Factor in Acne, J. Clin. Endocrinol. **1**:570 (July) 1941.

3. Reynolds, S. R. M., and others: Dermovascular Action of Certain Steroid Hormones in Castrates, Eunuchoid, and Normal Men, J. Clin. Endocrinol. **2**:228 (April) 1942.

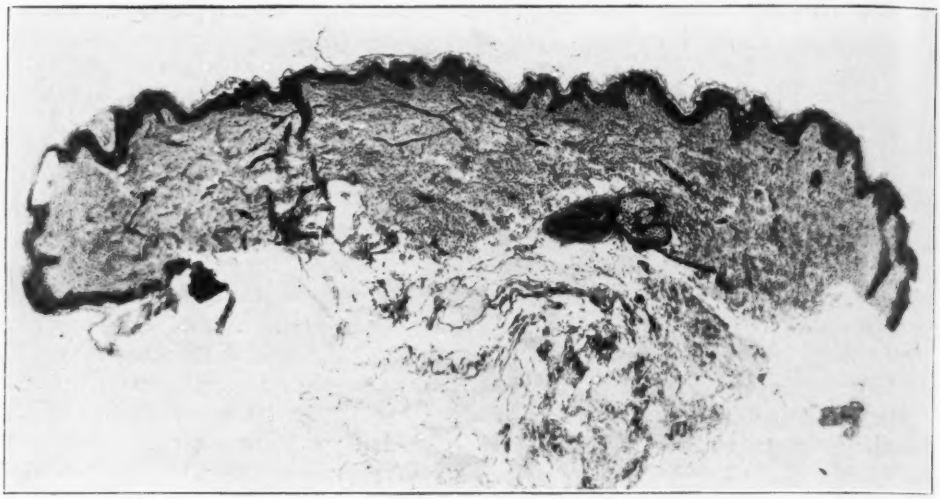


Fig. 1.—Section of normal skin from a boy aged 8 years. There is a single small sebaceous gland. Hematoxylin and eosin; × 48.

Fig. 2.—Section of skin from the same patient as in figure 1 sixteen days later. Sebaceous glands, hair follicles and coil glands are prominent. Note the increased thickness of the epidermis. Hematoxylin and eosin: × 48.

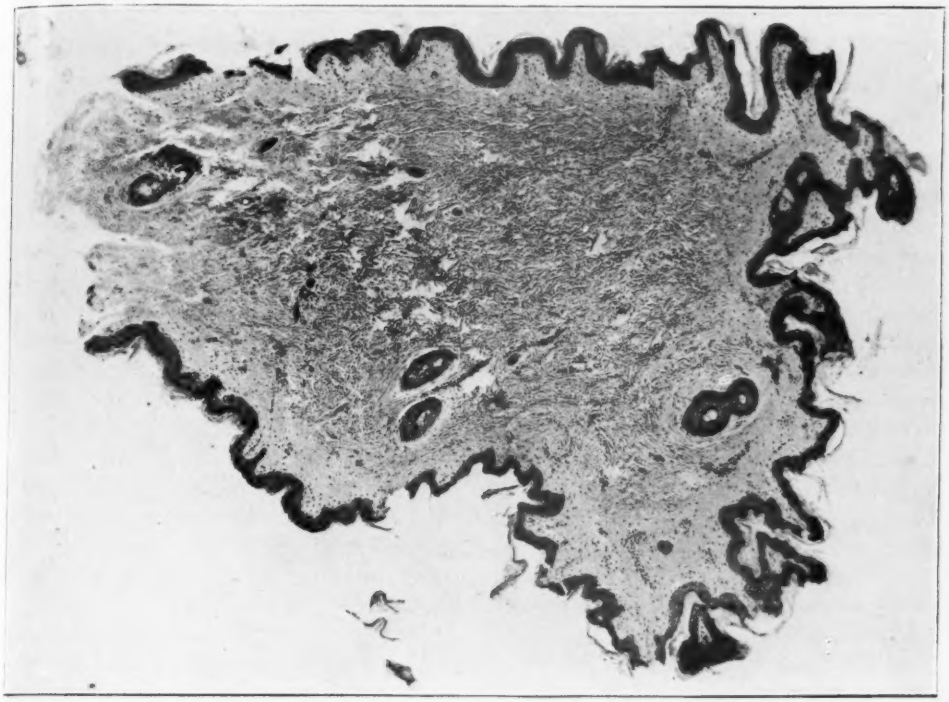


Fig. 3.—Section of normal skin from a boy aged 8 years. There are a few hair follicles but no sebaceous glands. Hematoxylin and eosin; × 48.

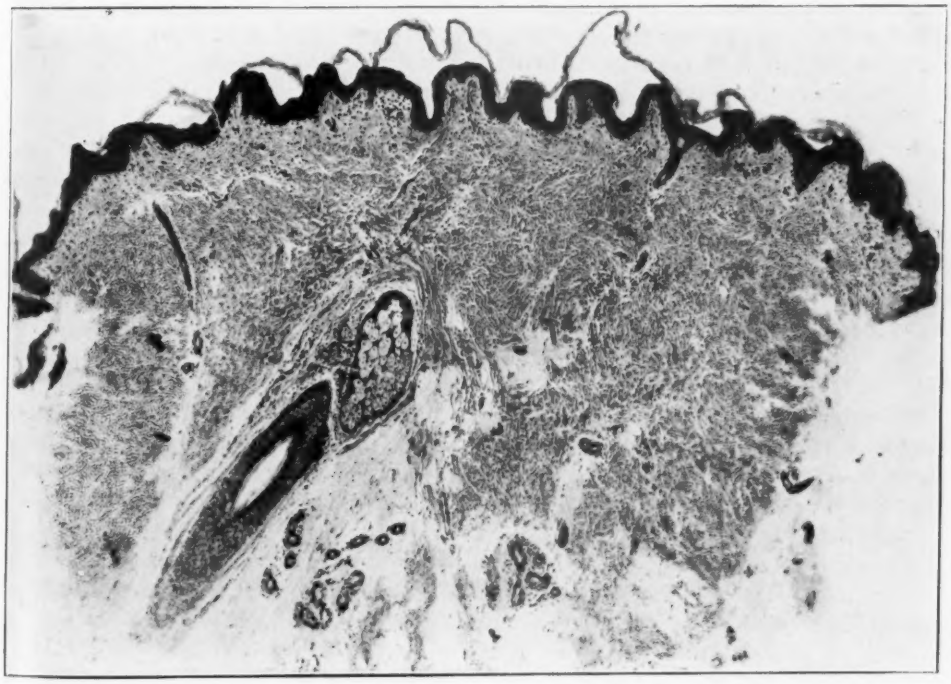


Fig. 4.—Section of skin from the same patient as in figure 3 sixteen days later. Note the large sebaceous gland, prominent hair follicles and numerous coil glands. Hematoxylin and eosin; × 48.

found in other structures of the skin, although in 3 of the 6 subjects the number and size of the hair follicles and sweat glands also showed a noticeable increase.

COMMENT

There has been considerable evidence of clinical nature, mostly in connection with observations on acne, suggesting that androgen exerts a stimulating effect on the sebaceous glands of the skin in man. J. B. Hamilton,[2b] reviewing earlier observations of others along with his own, pointed out that eunuchoid and castrated men and prepubertal boys—in other words, persons with known deficiency of androgen production—practically never acquire acne except when treated with large amounts of testosterone; moreover, acne thus produced in these subjects disappears soon after cessation of treatment and reappears with subsequent administration of testosterone. On the other hand, acne is not uncommon in women with virilism due to excessive production of androgen. It has been the belief of most dermatologists that acne is regularly associated with (and probably due to) excessive production of sebum by the sebaceous glands, and Hamilton concluded that the apparent role of androgen in the causation of acne is probably due to a stimulating effect on the sebaceous glands. A more direct evidence of such effect is, according to Hamilton, the "notable increase in secretion" on the surface of the skin of the nose and the appearance of "soft, white odoriferous material" on the preputial skin of patients treated with testosterone.

Our observations described here were already completed when Hooker and Pfeiffer [4] reported that the skin of their estrogen-treated rats became thin and the sebaceous glands were much reduced in size; however, animals receiving both estrogen and androgen were completely protected against these changes, and the sebaceous glands were even more numerous and larger than in the untreated animals. This again suggests that androgen may have a stimulating effect on the sebaceous glands, although the authors have admitted that their study throws no light on the action of androgen when administered alone.

We believe that our results constitute direct evidence of a stimulating action of androgen on the sebaceous glands in man. The question whether this is the only primary effect of the male sex hormone on the skin (stimulation of hair growth following automatically and secondarily) or whether activity of the hair follicle requires a further supply of androgen or of some other hormone remains open. With use of our direct approach it is planned to investigate in man some other pertinent questions, such as the effect of estrogen on sexual skin, the effect of testosterone on nonsexual regions of the skin and the effect of certain other endocrine substances suspected to be instrumental in hair growth.

SUMMARY

Six boys in the prepubertal age were treated parenterally with testosterone propionate for two weeks. Biopsies of the skin from the pubic region were made just before and two to three days after treatment. Decided increase in the number and size of the sebaceous glands was found in all subjects.

Dr. E. A. Oliver, Professor of Dermatology, Northwestern University Medical School, rendered valuable aid in this study.

The testosterone propionate used in these experiments was supplied by Roche-Organon, Inc., Nutley, N. J.

6 North Michigan Boulevard.

1 North Pulaski Road.

4. Hooker, C. W., and Pfeiffer, C. A.: Effects of Sex Hormones upon Body Growth, Skin, Hair and Sebaceous Glands in the Rat, Endocrinology **32**:69 (Jan.) 1943.

VITILIGO OF UPPER LIP IN PLAYERS OF WIND INSTRUMENTS

C. WENDELL FREEMAN, M.D.
AND
H. H. HAZEN, M.D.
WASHINGTON, D. C.

During the past three years one of us (C. W. F.) has observed 5 instances in which a member of a band who played some type of horn acquired vitiligo in the center of the upper lip. All of these patients were Negroes. Recently another eruption of this type was observed in a white trumpeter by one of us (H. H. H.).

The literature contains no similar observations, and the book on "Occupational Diseases of the Skin" by Schwartz and Tulipan [1] records none. In a personal interview Dr. Schwartz stated that no such instance had been reported, but that the condition might well come from a wooden mouth piece or from metal polish. A

Vitiligo of the upper lip in a player of a wind instrument.

careful history of our 6 cases revealed the fact that in each instance the mouth piece was of brass and no polish had been used on it. It is worthy of note that there was no preexisting history of any contact dermatitis.

All of the 6 patients were young men, 1 white and 5 Negro. All played a horn of some type, most of them the trumpet. The patients did not come to us because of the vitiligo, but for various other reasons. The patch of vitiligo was on the center of the upper lip and was never over ¾ inch (1.9 cm.) in diameter; its distribution corresponded to the area where the mouth piece rested on the lip. There was no macroscopic evidence of atrophy. A biopsy was not made. The patients were unanimous in their opinion that only those who made deep pressure with the mouth piece were affected. If an affected person stopped playing the instrument the area became smaller and some pigment returned.

1727 U Street Northwest.

1. Schwartz, L., and Tulipan, L.: A Text Book of Occupational Diseases of the Skin. Philadelphia, Lea & Febiger, 1939.

NECROBIOSIS LIPOIDICA DIABETICORUM WITH XANTHOMA CELLS

REPORT OF A CASE

LESLIE NICHOLAS, M.D.*
PHILADELPHIA

The eruption herein described occurred in a white man suffering from diabetes mellitus and pulmonary tuberculosis. The microscopic features of the lesions were so unusual that a complete description of the case is deemed to be worthy of publication. This is an attempt not to present a new clinical entity but rather to present material which may aid in the formation of a connecting link between necrobiosis lipoidica diabeticorum and the true lipoidoses.

REPORT OF CASE

J. R., a white man aged 34 years, was admitted to the Philadelphia General Hospital, division of tuberculosis, service of Dr. David Cooper, on Jan. 2, 1942.

History of Diabetes.—The diagnosis of diabetes was made in 1936, when the patient noted polyuria, weakness and loss of weight. The disease had been poorly controlled by diet alone until Feb. 12, 1938, when he sought treatment at the Philadelphia General Hospital for a rectal abscess. Studies at that time revealed no cutaneous changes and a completely normal physical condition, except for (1) a blood pressure of 104 mm. systolic and 60 mm. diastolic and (2) a rectal abscess. After immediate incision and drainage the latter healed rapidly. The hemogram showed 10,500 leukocytes per cubic millimeter, with 59 per cent neutrophilic polymorphonuclear leukocytes, 39 per cent lymphocytes and 2 per cent monocytes. The hemoglobin content was 14.5 Gm. per hundred cubic centimeters. The Kahn reaction was negative. On admission the urea nitrogen content of the blood was 10 mg. and the fasting blood sugar 440 mg. per hundred cubic centimeters; on subsequent determinations the sugar fluctuated from 390 to 61 mg. There were 224 mg. of total cholesterol and 128 mg. of cholesterol esters (or 57 per cent of the total) per hundred cubic centimeters of blood. The carbon dioxide–combining power of the blood plasma was 35 volumes per cent. Four days later, the carbon dioxide–combining power was 58 volumes per cent, and the total cholesterol content was 240 mg. and the content of cholesterol esters 166 mg. (or 69 per cent of the total). An electrocardiogram showed "T waves inverted in leads II and III and flattened in lead I; these findings are suggestive of some myocardial derangement." The fundi oculorum were normal. The patient was given a standardized daily diet composed of 70 Gm. of protein, 220 Gm. of carbohydrate and 100 Gm. of fat, together with 25 units of insulin (regular) a day (15-0-10). From the date of his discharge from the hospital, February 23, until his readmission, on Jan. 2, 1942, the diabetes was well controlled; the aforementioned diet was adhered to.

History of Cutaneous Lesions.—The patient noticed three lesions, one on the right arm and two on the left leg, during the latter part of the summer of 1941. Each began as a red papule and slowly increased in size. Gradually their color became more and more yellow. Trauma did not precede their appearance.

Family History.—The patient's mother was 70 years old and had suffered from mild diabetes for several years. No other metabolic and no neoplastic diseases were known to have occurred in the family, nor was there tuberculosis in any close relative.

Dermatologic Examination.—The skin was generally dry, thin and sallow. On the outer aspect of the right arm at the level of the insertion of the deltoid muscle, there was a circular, saucer-like depression with an infiltrated base measuring 1 cm. in diameter. The central portion was yellowish orange; the periphery, dark red. Numerous telangiectases coursed over

* Fellow in Dermatology and Syphilology, University of Pennsylvania School of Medicine.

From the Laboratory of Dermatological Research, University of Pennsylvania, and the Philadelphia General Hospital.

the entire lesion, excepting the peripheral zone. On the outer surface of the midzone of the left leg there were two slightly elevated, firm, canary yellow papules. Each measured 0.8 by 1 cm. and was surrounded by a narrow (3 to 4 mm.) reddish violet, scaly zone.

Remainder of Physical Examination.—The oral temperature was 99.6 F. and the pulse rate 110 and the respiratory rate 24 per minute. The blood pressure was 122 mm. systolic and 88 mm. diastolic. Other abnormal conditions observed were carious teeth, mild pyorrhea alveolaris and physical signs of infiltration in the apical portion of the left lung.

Laboratory and Special Studies.—The Kline reaction was negative. The fasting blood sugar was 360 mg. per hundred cubic centimeters; values on subsequent determinations during restandardization fluctuated between 280 and 81 mg., while the daily dose of insulin varied

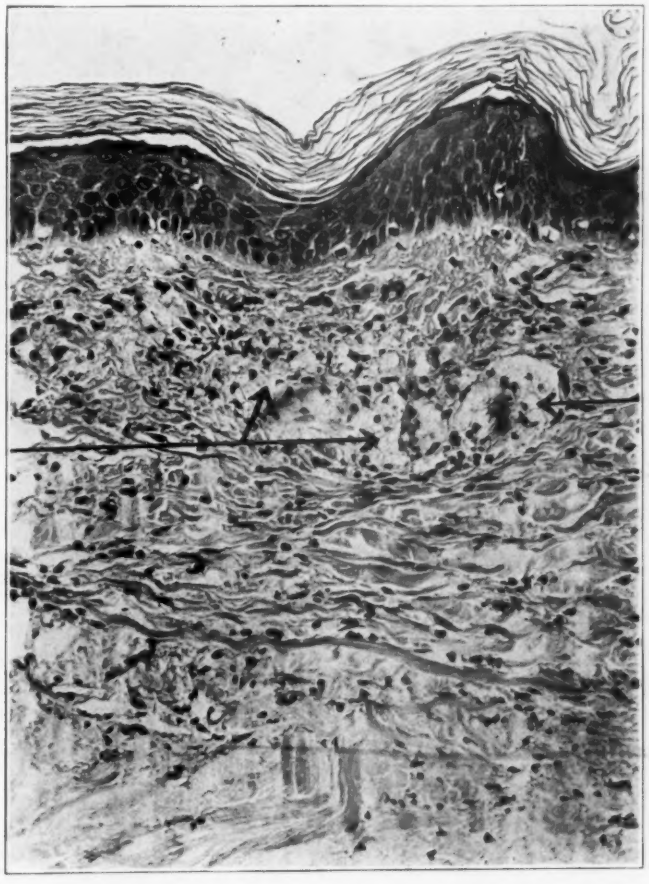


Fig. 1.—Foam cells in the upper part of the corium (marked by arrows). Hematoxylin and eosin; × 149.

between 48 and 64 units. The carbon dioxide–combining power of the blood plasma was 53 volumes per cent. The blood contained 19 mg. of urea nitrogen and 170 mg. of cholesterol per hundred cubic centimeters. The initial examination of sputum showed 30 acid-fast bacilli per hundred oil immersion fields; after the establishment of therapeutic pneumothorax repeated examinations of the sputum gave negative results. The first roentgenogram of the chest showed fibroexudative tuberculosis of the upper portion of the upper lobe of the left lung, small cavities of the fifth interspace posteriorly and spread to the upper lobe of the right lung. After therapy the picture showed an artificial pneumothorax, with 10 per cent collapse of the left lung, and numerous adhesions which prevented obliteration of the cavities. Peripheral vascular studies included (1) oscillometric readings, which were normal for both

legs and thighs but flat lines (zero) for both feet, and (2) a histamine test, which showed a delay in capillary response in both feet and legs, more definitely so on left side (i. e., the side with the dermatosis).

Clinical Course.—Artificial pneumothorax of the left side became successfully established. The diabetes was well controlled; the daily diet was 70 Gm. of protein, 130 Gm. of fat and 200 Gm. of carbohydrate. The insulin (regular and protamine zinc) requirement was 50 units (20-0-14 + 16 pz).

The distal lesion on the leg was excised for histologic study, and the wound healed uneventfully. Therapy was not attempted for the dermatosis.

More than a year later (February 1943) the patient was reexamined dermatologically. The lesion on the arm showed no change, and the site of the distal lesion on the leg was marked only by a narrow, pliable scar. The proximal lesion on the leg was remarkable because it had regressed to a considerable extent. It was no longer elevated or xanthochromic; it was merely an irregular, slightly scaly area of purplish brown pigmentation

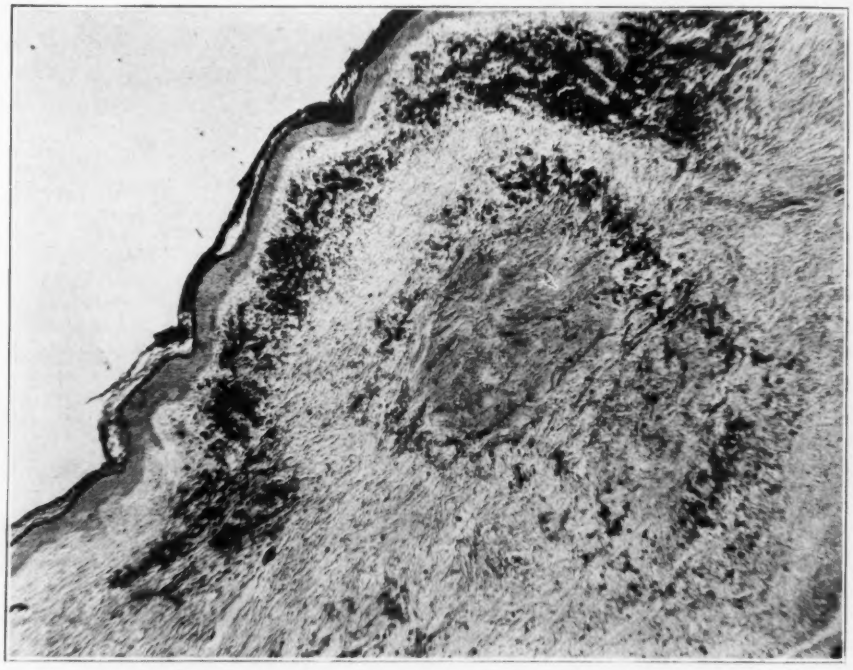


Fig. 2.—Section stained with sudan III and hematoxylin. The fat appears (1) under the epidermis in a black zone which consists largely of foam cells and (2) around and within an area of necrobiosis lower in the corium. The deeper gray shading in the center of the latter area is referable to extremely small fat globules. × 60.

which was more pronounced in the periphery. Only the slightest degree of atrophy and infiltration could be detected.

Microscopic Description.—The surface of the skin was fairly even. The epidermis was highly atrophic; the interpapillary pegs were obliterated. In sections stained with hematoxylin and eosin, foam cells in considerable numbers were found around the blood vessels in the subpapillary plexus. Deeper, the collagenous tissue appeared somewhat sclerotic, with definite broadening of the bundles at various places. In addition, some bundles were hugely broadened by what appeared to be edema and contained a few of the characteristic clefts which represent the presence of lipid crystals. Inflammatory features were not conspicuous, except in two sharply localized positions around blood vessels which were located closely adjacent to the focus of collagenous change; these were confined to an infiltration of lymphocytes. The larger blood vessels in the deeper parts of the corium exhibited moderate hyaline thickening of their walls and endothelial proliferation of the intima.

In a section stained with sudan III an abundance of fat appeared both within the foam cells and within the area of collagenous change, which had appeared to be edematous in sections stained with hematoxylin and eosin. In the foam cells, the fat appeared in typical form, cramming the cytoplasm of each cell to such an extent that the nucleus was obscured and the outline of the cell practically destroyed. Intermixed with the globules of fat there were numerous delicate acicular crystals which were doubly refractile when viewed with polarized light (crossed Nicol prisms).

In the deeper part of the corium, within the collagenous bundles that had undergone necrobiosis (they had appeared to be edematous in sections stained with hematoxylin and eosin), numerous excessively small fat globules were uniformly distributed. In the periphery of this necrobiotic focus the globules were larger. A few acicular crystals were present also in the necrobiotic area, but they varied as to double refraction with polarized light; small ones were anisotropic, but the long ones were not.

COMMENT

In an excellent review and report of 8 cases of necrobiosis lipoidica diabeticorum, Hildebrand, Montgomery and Rynearson [1] defined this unusual dermatosis as "a localized cutaneous lipoidosis, most often encountered in association with diabetes mellitus. It should be distinguished from the true xanthomas because of the absence of xanthoma or foam cells." Nonetheless, they cited the 3 cases in the literature in which xanthoma cells were reported. An analysis of the original reports of these 3 cases shows that 2 of them do not qualify as proved cases of this entity. In Feldman's [2] case, the distribution on the elbows and knees of a diabetic child aged 5 years could have been that of either xanthoma tuberosum or xanthoma juvenile in a diabetic person or of xanthoma diabeticorum. What necrosis there was was confined to the summit of the lesions (secondary necrosis perhaps traumatic) and was not reflected histologically in the corium in the form of necrobiosis. The diagnosis should have been any of these forms of xanthoma but not necrobiosis lipoidica diabeticorum. In the second case, Chargin's,[3] the patient "was not diabetic, but he certainly had necrobiosis lipoidica of the skin plus an additional phase in which xanthoma cells could be demonstrated." This case is not described formally in the literature, the cited statement appearing in a discussion by Peck of a clinical presentation. As a case of necrobiosis lipoidica diabeticorum it has shortcomings histologically, because, strangely, necrobiosis is not mentioned, although the xanthomatous changes are described with definiteness. However, on the face of it, it appears to be of the same order as my case but without diabetes. The "additional phase" to which Peck alluded will be discussed later in this paper.

Klaber's [4] case (the third) was similar indeed to the one herein reported. He demonstrated by means of the polarizing microscope a few doubly refracting crystals in the centers of the lipid foci and with appropriate staining "a marked degree of fatty infiltration, elastic dystrophy and a surrounding foam-cell and histiocytic infiltration." Klaber stated that each new lesion in his patient was preceded by irritation and observed that each passed at varying rates through several phases:

(1) Initially, a small fawn-coloured infiltrated papule.

(2) This increased in size and became violaceous in colour.

1. Hildebrand, A. G.; Montgomery, H., and Rynearson, E. H.: Necrobiosis Lipoidica Diabeticorum, Arch. Int. Med. **66**:851-878 (Oct.) 1940.

2. Feldman, S.: A Case for Diagnosis (Necrobiosis Lipoidica Diabeticorum? Xanthoma Diabeticorum with Necrosis?), Arch. Dermat. & Syph. **34**:326-328 (Aug.) 1936.

3. Chargin, cited by Peck in discussion on Feldman.[2]

4. Klaber, R.: Necrobiosis Lipoidica Diabeticorum: Report of a Case, Brit. J. Dermat. **46**:226-228 (May) 1934; Proc. Roy. Soc. Med. **27**:713-714 (April) 1933; **30**:976 (June) 1937.

(3) A raised, firm, yellow plaque developed in the center. At this stage the lesion strongly suggested an "inflamed" xanthoma.

(4) The firm, yellow plaque softened, and sometimes ulcerated, leading to a scanty, thick discharge. This ulceration might be omitted.

(5) Ultimately the lesions extended further in area, while flattening out. This extension ceased after a few weeks. At the final stage there was some yellowish white central atrophy, surrounded by an irregular scaly red or violaceous area of infiltration, with some marginal pigmentation.

In my patient it was impossible to trace the lesions through all of Klaber's five stages, but one can clearly assign the dermatosis (1) when originally noted by the patient to the first or papule stage, (2) when initially examined by me to the third or "inflamed xanthoma" phase and (3) when reexamined more than a year later to the fifth or final stage.

PATHOLOGY

Necrobiosis per se is a simple retrograde pathologic change which can usually be diagnosed with certainty in a section stained in the more or less routine manner, as with hematoxylin and eosin. It is only after the use of special stains for fat, though, that the entity necrobiosis lipoidica can be diagnosed with finality. In my case, the necrobiotic changes were somewhat atypical in that the collagenous bundles retained their normal architecture so well. There was no disruption of tissue and no evidence of an inflammatory reaction against the pathologic focus. But when stained with sudan III, this same area of swollen collagenous tissue had a rather uniform yellowish red color, imparted by numerous uniformly distributed, excessively small fat globules. This establishes with definiteness the diagnosis of necrobiosis lipoidica diabeticorum. The appearance was such as to suggest an imbibition of fat by the collagenous tissue.

Next, as to the foam cells. Their presence in Klaber's case and in mine should not be construed as vitiating the accepted tenets concerning the pathogenesis of necrobiosis lipoidica diabeticorum. The pathogenesis of the lipoid changes in the necrobiotic areas remains the same in all cases, as judged by histologic studies; at least, the appearance is the same. After that, the foam cells are probably only incidental and secondary developments in connection with a focus of lipoid saturation (Peck's "additional phase" already referred to). That is, the histiocytes surrounding the focus imbibe the superabundant lipid developed within it.

Why, next, does such imbibition occur so rarely in necrobiosis lipoidica diabeticorum? Why did it occur in my case and in Klaber's and not in others? Is it due to more or less accidental conditions of the fat diet or of the fat metabolism, or have histiocytes become specially "adapted," for whatsoever special reason, in the direction of imbibition of lipid? These questions must be left unanswered in this paper, except to note that the latter subject has been touched on by Weidman [5] in connection with "essential or intrinsic xanthomatosis." In any event, the occurrence of foam cells in a zone lying more or less around a focus of necrobiosis lipoidica diabeticorum should interpose only a temporary obstacle during the differential diagnostic considerations of a histologic section.

SUMMARY

A case of necrobiosis lipoidica diabeticorum containing xanthoma cells in a man suffering from diabetes mellitus and pulmonary tuberculosis is reported. It is probably the second valid instance of such a combination. The histamine test

5. Weidman, F. D.: Xanthosarcoma of the Cheek Succeeding Xanthosarcoma of the Forearm: Multiple Tumors Versus Metastasis, Arch. Surg. **34**:792-828 (May) 1937.

showed a delayed capillary response in the affected limb. Doubly refracting crystals were demonstrated among the foam cells and in the necrobiotic areas.

NOTE.—Since the completion of this communication, another report [6] of the presence of foam cells in necrobiosis lipoidica diabeticorum has appeared. In this instance, the spatial relationship between the foam cells and the necrobiotic tissue was not indicated and anisotropic crystals were not described.

The sections are filed in the Laboratory of Dermatologic Research, University of Pennsylvania, accession no. 4133, and at the Army Medical Museum, accession no. 91224.

36 Hamilton Walk.

6. Bernstein, E. T.: Necrobiosis Lipoidica Diabeticorum, Arch. Dermat. & Syph. **47**: 602-603 (April) 1943.

DERMATITIS DUE TO NAIL POLISH

REPORT OF TWO CASES, WITH EXPERIMENTAL STUDIES

HARRY KEIL, M.D.
NEW YORK
JOSEPH J. RUSSO, M.D.
ALBANY, N. Y.
AND
LAIRD S. VAN DYCK, M.D.
NEW YORK

In a recent review of the subject of dermatitis due to nail polish, Osborne and his co-workers [1] detailed their clinical observations in 100 cases of this disease encountered by them since 1926. They stressed the increasing incidence in the past three years. It is probable that the frequency of nail polish dermatitis can now be counted in the hundreds, if not in the thousands. Because the female population is being exposed to such mass sensitization (occasional examples are also encountered in men using colorless nail polish), the subject has become important. Simon [2] found that the cause of this dermatitis is a "formaldehyde-sulfonamide resin."

The protocols of the following 2 cases provide data bearing on some points of fundamental interest. Among these are: 1. Does nail polish cause a generalized hypersensitivity of the skin? 2. If such generalized hypersensitivity of the skin occurs, does it involve all parts of the body? 3. Why are so few patients seen with lesions on the fingers, the areas closest to the applied sites of nail polish? Osborne and his co-workers noted only 2 instances out of 100 in which lesions appeared on the hands and fingers, and other observers, including ourselves, have seen an eruption in this localization in only exceptional instances.

REPORT OF CASES

CASE 1.—A woman aged 36 years had a rash on the eyelids and neck about eight months before she came to the clinic. This attack lasted a week. Six weeks before the present observation, the eruption recurred.

On examination the skin of the eyelids was erythematous, swollen and thickened. There were a few scaly areas on the forehead and cheeks, with discrete lesions on the sides of the neck. The diagnosis of dermatitis due to nail polish was readily made.

As we discussed the matter with the patient, she recalled that at times, particularly on Sundays, the eruption became worse. It was her practice to apply the polish on Saturday, and this had been done on the afternoon of the preceding Saturday. That evening the condition of the eyelids flared up. She had used nail polish for some six years and had purchased a fresh supply about four months previously, but it was her practice to switch from one to another of at least six different polishes.

Patch tests with six different nail polishes, including a colorless type used for securing better adhesion, were applied. These were applied to the chest, the right arm and the right thigh. In forty-eight hours all the areas showed positive reactions to all six preparations.

From the Skin and Cancer Unit, New York Post-Graduate Medical School and Hospital, Columbia University.

1. Osborne, E. D.; Jordan, J. W., and Campbell, P. C.: Dermatitis Venenata Due to Nail Lacquer, Arch. Dermat. & Syph. **44**:604 (Oct.) 1941.

2. Simon, F. A.: Nail Polish Eczema, South. M. J. **36**:157 (Feb.) 1943.

The reactions on the chest were somewhat more intense than those on the arm and thigh. These reactions varied from red and swollen areas (1 to 2 plus) to more intense responses with a definite, though mild, vesicular component (2 to 3 plus).

The skin of the left side of the neck was then painted with the nail polish to which the strongest reaction had been obtained, and this was allowed to dry. In addition, patch tests were applied to the dorsal aspect of the proximal portion of the left fourth finger and to the left palm and sole; these were, of course, applied under occlusive coverings. That evening the patient had to remove the nail polish put on the skin of the neck, owing to an intense local reaction, and the next morning the eyelids were swollen (direct contact? focal reaction?). The patch tests, which were removed in forty-eight hours, revealed an edematous and erythematous reaction on the finger, the intensity being somewhat less than the responses obtained on other parts of the body. The sites of the patches on the palm and sole showed no abnormalities.

A few days later the skin of the adjacent finger was painted in an area corresponding to that on the left fourth finger, but this time the nail polish was allowed to dry in situ without an occlusive covering. Forty-eight hours later this area was red and slightly swollen. The intensity of this response was less than that observed on the skin of the left fourth finger, which had been covered with an occlusive patch.

The results of the patch tests showed conclusively that the skin of this patient was generally hypersensitive to all six nail polishes. This hypersensitivity included the skin of the dorsa of the fingers, whereas the palm and sole were unaffected even after an exposure of forty-eight hours under an occlusive patch. The application of a nail polish to the integument of a finger showed that with adequate exposure, whether covered or uncovered, a positive reaction could be obtained, although the intensity was greater under an occlusive covering.

The results also showed that irrespective of the manner in which the polish was applied, whether directly on skin or indirectly by first painting a piece of linen, the results were essentially similar, provided contact with skin was adequate. If there were any important differences, we failed to detect them. This conclusion was substantiated by patch tests made on many other patients afflicted with this dermatosis.

CASE 2.—A woman 30 years old had a red scaly eruption on the chin and cheeks, with vesicular elements superimposed on the ears. This had been present for the past two years. The upper eyelids were only slightly affected at this time.

She had used nail polish, nail polish remover and cuticle remover for fifteen years. Of the more recently used nail polishes, one had been applied for three years, another for the past week and a third, a colorless coating used as a base for other nail polishes, for the past two months.

Patch tests were made with the two colored nail polishes, the nail coating, the cuticle remover and the nail polish remover. The patches were applied to both the upper part of the back and the right thigh. The positive reactions were essentially the same in the two regions, although slightly weaker on the thigh. The coating elicited a large edematous area with some vesicles (a 3 to 4 plus reaction); the two colored nail polishes each gave edematous and vesicular reactions (each 3 plus), and the remaining substances produced no reactions.

The patient then applied one of the colored nail polishes to the dorsum of each of three adjacent fingers on the right hand (third, fourth and fifth fingers) in a small area between the metacarpophalangeal joint and the proximal phalangeal joint. The nail coating was applied similarly on the dorsal aspect of the same fingers in areas between the distal phalangeal joint and the margin of the nail. The polishes were permitted to dry in situ. The usual washing of the hands was permitted, since washing alone does not remove the adherent film. The films of dried polish were removed from the fifth finger after six hours with the use of nail polish remover, from the fourth finger after twelve hours and from the third finger after twenty-four hours. It must be noted that these were not applied as patch tests under occlusive coverings but that every effort was made to duplicate the actual conditions of exposure.

In the areas exposed to colored nail polish and nail coating for six hours, there were no reactions at either site. In the areas painted for twelve hours, the colored nail polish elicited a positive reaction (edema and redness), whereas the site of the nail coating was unaffected.

In the areas exposed for twenty-four hours, the colored nail polish produced a more intense edematous response and the reaction to the coating was also faintly positive (slight redness and edema).

At this time, the patient, being not entirely convinced about the diagnosis, applied another nail polish to her nails. The next morning there was a relapse of the eruption with intense involvement of the upper eyelids. She is now convinced.

The most intense reactions on the back and thigh were elicited by the nail coating. It seems fair to assume that, of all the nail polishes used, the patient was most hypersensitive to the nail coating in these areas of the body. Contrariwise, this coating elicited less reaction on the distal portions of the fingers than did the colored nail polish on the proximal portions of the phalanges. From these data we are inclined to conclude that the distal portions of the fingers were less hypersensitive than the proximal portions of these fingers. Perhaps this difference in response may be explained on anatomic grounds. In any event, the reactions displayed by the skin of the fingers were far less intense than those shown by the skin of the back and thigh.

COMMENT

If it is generally true, and case 1 would support this view, that the reactions of the skin of the fingers are less intense than those of the skin of the back and thigh, it would seem that, contrary to other forms of contact dermatitis, the hypersensitivity to nail polish is induced more readily at points remote from the site of application. However, this conclusion is more apparent than real, because anatomic factors enter into consideration. It must be borne in mind that the skin of the eyelids, face and neck is thinner than that of the fingers and more readily sensitized by contact. In case 1 it was shown that simple painting of the skin of the neck was sufficient after a short exposure to cause a local and, possibly, a focal reaction in the eyelids (?). In case 2 it was observed that an exposure time of twelve hours was required to provoke a mild reaction on the proximal part of the dorsal aspect of the phalanges, whereas twenty-four hours was needed for similar manifestations to appear on the distal portions of the same fingers. It must be noted that this temporal relation held only for nail polish applied to the skin without any impermeable covering.

This difference in reaction time is important because any nail polish that is carried to the fingers accidentally during the application to the nails or otherwise, particularly when the polish is colored, is apt to be removed by the patient using nail polish remover. It was shown in case 2 that, even with ordinary washing of the hands after the polish had dried on the skin, a reaction was produced in the skin after a certain exposure time, which ranged from twelve to twenty-four hours. In other words, the skin of the finger will respond clinically to nail polish provided there is sufficient exposure. It is possible that in some cases the reactions on the fingers are so mild as to be overlooked easily. Contrariwise, if such polish should contaminate accidentally the palms or soles, it seems most unlikely that lesions would be caused, owing to the unusually thick horny layer in these parts. Thus, in case 1 patch tests with a nail polish to which the patient was hypersensitive, produced negative reactions on the palm and sole, even when covered by an occlusive patch.

SUMMARY AND CONCLUSIONS

Two cases are reported in which patch tests indicated a generalized hypersensitivity to nail polish.

This generalized hypersensitivity did not include such areas of the body as the palms and soles, owing probably to anatomic factors, such as an unusually thick keratin layer.

Lesions on the fingers resulting from hypersensitivity to nail polish are not often seen, because an exposure time of about twelve to twenty-four hours is needed. This requirement is usually not met, owing to the relatively prompt removal of nail polish from these areas with polish remover. If adequate contact is established for this length of time, clinical lesions can be produced, and our 2 cases offer experimental proof for this view.

The thin skin of the eyelids and neck is apparently more readily sensitized than the thicker epidermis of the fingers. An important element is the duration of exposure, which is apt to be greater in the case of the neck and face than of the hands.

Patch tests under an occlusive covering generally give more intense reactions than simple application of nail polish without an occlusive cover. Either method can be used. We prefer the patch tests, because the more intense reactions can be better evaluated.

It is immaterial, in our view, how the patch test is applied to the skin, whether by painting the nail polish directly on to skin or indirectly through the medium of a piece of linen; the only requisite is adequate contact with the skin.

509 Madison Avenue.
343 Madison Avenue.
206 East Seventeenth Street.

PRODUCTION OF BULLAE IN THE SKIN OF THE DUCK

II. EXPERIMENTS WITH BULLAE PRODUCED BY VESICANTS

LEON GOLDMAN, M.D.

NORTON NELSON, PH.D.

AND

I. ARTHUR MIRSKY, M.D.

CINCINNATI

In a previous communication evidence was presented which demonstrated that the local application of various irritants can produce large collections of fluid in the skin of ducks.[1] This observation made possible the study of cutaneous collections of fluid in an experimental animal which is readily available.

With the use of the duck, it has been possible to study various dermatologic phenomena. Since the studies could not be continued, it was deemed advisable to present some of our preliminary data in order to demonstrate the value of the aforementioned phenomena in experimental dermatology.

The first series of experiments consisted in the determination of the minimal dose of a vesicant necessary to induce blisters in a series of ducks. It was observed that with bis-beta-dichlorodiethylsulfide (mustard agent) the minimal vesicant dose varies somewhat with the age of the animal. In a series of ducks aged 16 months and over, 0.017 mg. of mustard agent produced localized collections of fluid in the skin, whereas the skin of a series of ducks of ages from 2 to 6 months did not react until a dose of 0.08 mg. was applied. Thus, the younger birds required a greater dose than did the older birds for the production of similar cutaneous lesions.

The skin of man is known to become sensitized to mustard agent. Therefore, it became of interest to find whether the same phenomenon can occur in the duck. Toward this end the skin of a series of ducks was treated daily with applications of subminimal effective doses of mustard agent. After an interval of from five to eight days, lesions were produced with subminimal effective doses. This suggests that the skin of the duck, like that of man, can become sensitized to mustard agent.

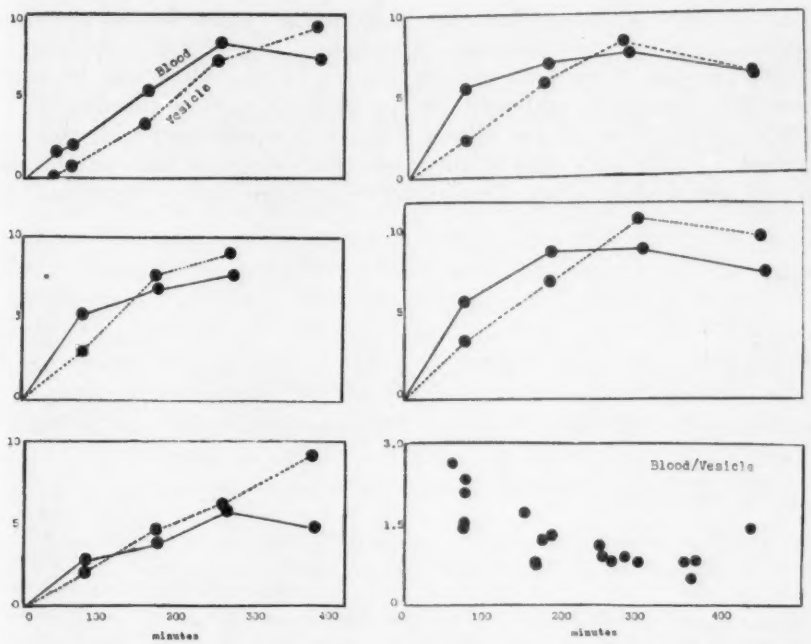
The influence of local and general anesthesia on the production of blisters was studied in a group of ducks. Preliminary studies revealed that the sensitivity of the skin to mustard agent decreased when the bird was kept under pentobarbital sodium anesthesia. Thus, the application to the skin of an effective dose of mustard agent was frequently ineffective in the anesthetized bird. Only a partial inhibition of the vesicant effect of mustard agent was observed when the agent was applied to a locally anesthetized area of skin. In the human skin, local anesthesia produced no inhibition of vesiculation.

From the May Institute for Medical Research, the Jewish Hospital.

1. Mirsky, I. A., and Goldman, L.: The Production of Bullae in the Skin of the Duck: Preliminary Report, Arch. Dermat. & Syph. **48**:161-163 (Aug.) 1943.

Attempts to influence the development of blisters in the skin of ducks by means of administration of adrenal cortex extract, iontophoresis with mecholyl chloride or injections of solution of posterior pituitary were without success.

Experiments were performed on the fluid of the blister produced by lewisite, chlorovinyldichloroarsine. A series of injections of trypan blue were made, and it was observed that this dye penetrated rapidly into the fluid collection in the skin if the dye was injected intravenously soon after the lewisite was applied to the skin. However, once the bullae had been produced, the dye no longer accumulated in them unless the lesions were punctured and the contents drained. Then, when the fluid reappeared the dye was present in it. If the cutaneous lesions were older than three days when the trypan blue was injected for the first time, it did not accumulate in the blisters.



Levels of free sulfathiazole in the blood and in vesicles of ducks after oral administration of the drug. Vesicles produced by applications of lewisite (chlorovinyldichloroarsine) to skin.

The relation between the sulfathiazole content of blister fluid and that of blood was studied in a series of birds. After blisters were produced by application of lewisite on the skin, sulfathiazole was administered by mouth. Then, the concentrations of free sulfathiazole in the whole blood were compared with the concentrations of sulfathiazole in the blisters. During the rising phase of the sulfathiazole curve of the blood, a lag occurred in the sulfathiazole curve of the blister fluid. With the stabilization and consequent fall in the drug level of the whole blood, the lag persisted so that concentration of sulfathiazole was then higher in the blister fluid than in the blood (figure). Comparison between blister fluid and blood plasma revealed that the sulfathiazole level of the fluid was in equilibrium with that of the plasma.

A similar study was performed on a patient with extensive bullous dermatitis herpetiformis, and similar data were obtained. The following data from such a patient are illustrative:

	Blood, Mg. 100 Cc.	Blister Fluid, Mg. 100 Cc.
Sulfathiazole (free)	3.8	2.2
Sulfathiazole (conjugated)	3.3	2.5
Sulfadiazine	5.6	6.0
Sulfadiazine	5.6	6.2
Sulfapyridine	2.4	1.8

It is probable from the preceding data that blister fluid is in equilibrium with the blood plasma and that a rapid interchange of diffusible constituents occurs.

SUMMARY

Large collections of serous fluid can be produced in the skin of the duck by local applications of suitable quantities of vesicants. By repeated applications of these agents, the skin of the duck can become sensitized. Fluid may be aspirated from these fluid collections and used for the study of various constituents. The sulfonamide concentration of the blister fluid is in equilibrium with that of the blood plasma. Preliminary experiments show that the duck may be of value in experimental dermatology when the production of large blisters in animal skin is desired.

COMPLEMENT FIXATION TEST FOR LYMPHOGRANULOMA VENEREUM

RESULTS OBTAINED WITH ITS USE

ARTHUR W. GRACE, M.D.
BROOKLYN
AND
GEOFFREY RAKE, M.B., B.S.
NEW BRUNSWICK, N. J.

The earliest report of the employment of a complement fixation test for the diagnosis of lymphogranuloma venereum was made by Kitchevatz and Kitchevatz-Petrovitch.[1] In this and in subsequent papers,[2] specific results were claimed for the use of an antigen consisting of an emulsion of a gram-positive, non-acid-fast, spore-bearing bacillus isolated from a lymphogranulomatous inguinal bubo. Schmidt-LaBaume,[3] after commenting on the indefinite and inconclusive results obtained by Frei and Musger in complement fixation studies of lymphogranuloma venereum, reported for the first time on the use of antigens prepared from lymphogranulomatous pus. The early results were encouraging, but later ones were so uncertain as to cast doubt on the procedure. Löhe and Blümmers [4] could not demonstrate complement-fixing antibodies with antigens prepared from lymphogranulomatous nodes or pus. Nicolau and Banciu [5] failed similarly with such pus. Coutts and his co-workers [6] claimed success with an antigen made by extracting lymphogranulomatous pus or nodes with isotonic solution of sodium chloride, but Banciu [7] was unable to confirm their conclusions, and Prats [8] published data indicating that the test of Coutts was not reliable. Antigens prepared from diluted and filtered lymphogranulomatous pus showed a moderate degree of specificity when used by

From the Long Island College Hospital and the Department of Dermatology and Syphilology, the Long Island College of Medicine, Brooklyn, and the Squibb Institute for Medical Research, New Brunswick, N. J.

1. Kitchevatz, M., and Kitchevatz-Petrovitch, V.: Contribution à l'étiologie de la maladie de Nicolas-Favre. Etude bactériologique et réaction de fixation du complement, Bull. Soc. franç. de dermat. et syph. **34**:879, 1927.

2. Kitchevatz, M., and Kitchevatz-Petrovitch, V.: Recherches bactériologiques et sérologiques dans la poradénite, Bull. Soc. franç. de dermat. et syph. **35**:51, 1928; Nouveau résultats de la réaction de fixation dans la maladie de Nicolas-Favre, ibid. **35**:459, 1928.

3. Schmidt-LaBaume: La valeur diagnostique de la réaction de fixation du complement dans la lymphogranulomatose inguinale, Bull. Soc. franç. de dermat. et syph. **38**:562, 1931.

4. Löhe, H., and Blümmers, K.: Weitere Mitteilungen über die Lymphogranulomatosis Inguinalis, Med. Klin. **27**:614, 1931.

5. Nicolau, S., and Banciu, A.: Lymphogranuloma inguinale, Ann. de dermat. et syph. **3**:332, 1932.

6. Coutts, W. E.; Landa Perroni, F., and Martini, J.: Lymphogranuloma inguinale-Antigen als Rezeptor in der Wassermannschen Komplementbindungsreaktion, Dermat. Wchnschr. **98**:558, 1934. Coutts, W. E., and Ponce, T.: The Complement Fixation in the Diagnosis of Lymphogranulomatosis Venerea, J. Lab. & Clin. Med. **20**:629, 1935.

7. Banciu, A.: Untersuchungen über die Komplementbindungsreaktion bei Lymphogranuloma Inguinale, Zentralbl. f. Haut- u. Geschlechtskr. **52**:629, 1936.

8. Prats, F.: Reaccion de desviacion del complemento, hemoreaccion de Ravaut y examen de fundo de ojo, como medios diagnósticos en la linfogranulomatosis inguinal benigna (enfermedad de Nicolàs y Favre), Rev. méd. de Chile **63**:298, 1935.

Melczer and Sipos [9] but none at all when used by Midana.[10] It is now believed that failure to demonstrate complement-fixing antibodies in the serums of lymphogranulomatous persons by the use of pus antigens was due to the low concentration of virus in such pus. The growth of virus in high titer in the yolk sac of the developing chick embryo [11] has enabled antigens to be prepared containing any desired concentration of virus. Reports on the successful use of such antigens have already been made.[12]

In this paper are presented discussions of the following subjects: a comparison of the results obtained with the complement fixation test and the Frei test on 202 lymphogranulomatous and nonlymphogranulomatous persons; the relation between the titer of the serum and the dimensions of the Frei reaction; the titer of the serum in symptomatic and asymptomatic lymphogranuloma, and the effect of the administration of sulfonamide compounds on the titer of the serum.

MATERIALS AND METHODS

The antigen used was a suspension in isotonic solution of sodium chloride of the elementary bodies of the virus, freed from inert material by differential centrifugation.[12a,b] Other antigens, prepared by treating the infected yolk sac suspensions with ether or urea, have been described recently [13] and give highly satisfactory results.[14] The concentration at which the antigen is employed in the test is determined by prior titration with known lymphogranulomatous serums. In the performance of the test a mixture of 0.2 cc. of each of the following materials, added in the order given, is allowed to react for seventy-five minutes at 37 C.; patient's serum, inactivated at 56 C. for one-half hour and diluted 1 in 2 or more; 2 hemolytic units of guinea pig serum complement, and antigen. The presence or absence of fixation is then determined by the addition of a 3 per cent suspension of sensitized sheep's red corpuscles and further incubation for thirty minutes at 37 C. As in the Wassermann test, a positive reaction is indicated by the absence of, and a negative reaction by complete, hemolysis of the sheep's corpuscles. Adequate controls are employed.

In this study the majority of the serums were tested in a series of final dilutions beginning with 1 in 6 and 1 in 15 and thereafter increasing twofold up to 1 in 1,920. Serums from normal persons rarely fixed complement in a dilution as high as 1 in 6. All serums, therefore, which regularly fixed complement in this dilution (1 in 6) or higher, were reported as giving a positive reaction. Frei tests were performed on all persons who

9. Melczer, N., and Sipos, K.: Komplementbindungsversuche bei Lymphogranuloma inguinale, Arch. f. Dermat. u. Syph. **176**:176, 1937.

10. Midana, A.: Richerche sulla reazione di deviazione del complemento nella malattia di Nicolas e Favre, Gior. ital. di dermat. e sif. **79**:859, 1938.

11. Rake, G.; McKee, C. M., and Shaffer, M. F.: Agent of Lymphogranuloma Venereum in the Yolk-Sac of the Developing Chick Embryo, Proc. Soc. Exper. Biol. & Med. **43**:332, 1940.

12. (*a*) McKee, C. M.; Rake, G., and Shaffer, M. F.: Complement Fixation Test in Lymphogranuloma Venereum, Proc. Soc. Exper. Biol. & Med. **44**:410, 1940. (*b*) Rake, G.; Shaffer, M. F.; Grace, A. W.; McKee, C. M., and Jones, H. P.: New Aids in the Diagnosis of Lymphogranuloma Venereum, Am. J. Syph., Gonor. & Ven. Dis. **25**:687, 1941. (*c*) Shaffer, M. F.; Rake, G.; Grace, A. W.; McKee, C. M., and Jones, H. P.: Lymphogranuloma Venereum Intercurrent with Other Venereal Diseases, ibid. **25**:699, 1941. (*d*) Shaffer, M. F.; Rake, G., and Grace, A. W.: Yolk Sac Antigens in the Diagnosis and Epidemiology of Lymphogranuloma Venereum, ibid. **26**:271, 1942.

13. Nigg, C.: Action of Urea and Ether on Agent and Complement-Fixing Antigen of Lymphogranuloma Venereum, Proc. Soc. Exper. Biol. & Med. **49**:132, 1942.

14. Phenolized antigens which have recently been described (Nigg, C., and Bowser, B. M.: Enhancement with Phenol of the Serological Reactivity of Lymphogranuloma Venereum Antigens, Proc. Soc. Exper. Biol. & Med., to be published) offer advantages in greater clarity and stability, in the higher titers of antigen which can be obtained and in the apparent absence of nonspecific reactions which both other antigens had shown with serums from patients with early syphilis.

furnished serums for the complement fixation test. Standardized yolk sac material (lygranum) was employed as the antigen in the cutaneous test, in which the presence of a papule 6 by 6 mm. or larger in diameter at the end of forty-eight to ninety-six hours after the performance of the test was regarded as a positive reaction. Control material prepared from normal yolk sacs was also used in every cutaneous test.

The clinical material consisted of 202 persons who presented the conditions listed in table 1 and who were either inpatients or outpatients of the New York Hospital or the Long Island College Hospital.

TABLE 1.—*Distribution of Clinical Conditions Among 130 Subjects with Positive and 72 with Negative Reactions to the Complement Fixation Test for Lymphogranuloma Venereum*

130 Positive Reactors		72 Negative Reactors	
Clinical Condition	Number of Subjects	Clinical Condition	Number of Subjects
Proctitis with stricture	32	Congenital syphilis	28
Proctitis without stricture	21	Cystitis	16
Inguinal adenitis	16	Latent syphilis	7
Latent syphilis	16	Primary syphilis	3
Primary syphilis	9	Hemorrhoids	3
Cystitis	7	Abacterial urethritis	2
Fistula in ano	6	Rectal polyp	2
Abacterial urethritis	5	Penile ulcer	2
Perianal fissure and inflammation	4	Fistula in ano	1
Penile ulcer	2	Proctitis without stricture	1
Rectal polyp	2	Ulcerative colitis	1
Penile scars, cured gonorrhea	1	Carcinoma of rectum	1
Perirectal abscess	1	Anal fissure	1
Ulcer on labium majus	1	Pruritus ani	1
Fibrosis of corpora cavernosa	1	Tuberculous abscess of labium majus	1
Periurethral abscess and chronic prostatitis	1	Mass in breast	1
Chancroid	1	Psychoneurosis	1
Gonorrhea	1		
Penile ulcer with inguinal adenitis	1		
Nongonococcic urethral stricture	1		
Contact with lymphogranulomatous person	1		

TABLE 2.—*Comparison of Results Obtained with the Frei Test and with the Complement Fixation (C. F.) Test for Lymphogranuloma Venereum*

Results	Number of Subjects	Percentage
Both tests positive	105	52.0
Both tests negative	68	33.6
Frei test negative, C. F. test positive	21	10.4
Frei test doubtful, C. F. test positive	4	2.0
Frei test positive, C. F. test negative	3	1.5
Frei test doubtful, C. F. test negative	1	0.5

USE OF COMPLEMENT FIXATION AND CUTANEOUS TESTS IN LYMPHOGRANULOMATOUS AND IN NONLYMPHOGRANULOMATOUS PERSONS

The complement fixation reaction was positive in the serums of 130 and negative in those of 72 persons. A comparison of the results obtained by the use of cutaneous and serologic tests is given in table 2.

Positive results were obtained with both tests in 105 persons, all of whom either presented manifestations which could be regarded as lymphogranulomatous or had then or had had in the past another venereal disease. It has already been shown

that a considerable proportion of persons who have or have had gonorrhea, chancroid or acquired [12c] (but not congenital [15]) syphilis have a positive Frei reaction and a positive complement fixation reaction; this can be explained most satisfactorily by the hypothesis that lymphogranuloma, in common with syphilis, can exist in an asymptomatic form. It was felt, therefore, that the positive reactions obtained in this group were due to symptomatic or asymptomatic infection with the lymphogranuloma virus. Twenty-five persons had a complement fixation reaction that was positive and a Frei reaction that was either negative or doubtful. The clinical manifestations and histories of this group were then reviewed, in order to determine the likelihood of the existence of lymphogranuloma venereum among them. It was found that 9 had latent and 1 primary syphilis, 1 prostatitis and periurethral abscess, 1 postgonorrheal urethral strictures, 1 scarring of the penis and cured gonorrhea, 1 chancroid, 1 penile sore and inguinal adenitis, 1 healed inguinal adenitis, 1 nongonococcic urethral stricture, 1 abacterial urethritis, 3 cystitis, 2 rectal polypi and 2 perianal fissure. More than one half of the group was, therefore, composed of persons with venereal diseases other than symptomatic lymphogranuloma venereum, and the remainder presented clinical conditions which could be regarded as lymphogranulomatous. In view of the frequent coexistence of lymphogranuloma and acquired venereal disease and the presence of a positive complement fixation reaction, it was decided to regard all of the group as showing symptomatic or asymptomatic lymphogranuloma venereum. Of the 3 persons whose reaction to the Frei test was positive and to the complement fixation test was negative, 1 had early syphilis, 1 latent syphilis and 1 acute cystitis. Since in certain cases acute cystitis is undoubtedly due to lymphogranuloma venereum, there was no reason to believe that the results obtained with the cutaneous test in this group were nonspecific. The total number of persons, therefore, who had either positive complement fixation or a positive Frei reaction or both and who could have been suffering from symptomatic or asymptomatic lymphogranuloma venereum was 133, of whom 97.7 per cent reacted positively to the former and 81.2 per cent to the latter test. We are of the opinion that these percentages represent the relative degrees of sensitivity of the two tests. Whether or not false negative reactions occur with either test in persons who present lesions which could be regarded as lymphogranulomatous (such as many in the right half of table 1) can be answered only by the concurrent use on the same subject of these tests, together with others capable of demonstrating the presence of the lymphogranuloma virus in the lesions. Normal persons show few if any false positive reactions to the complement-fixation test,[12b] and all the serologic evidence at present available points to the specific nature of this test. However, as has been shown elsewhere, not only is the agent of lymphogranuloma venereum morphologically similar to those of meningopneumonitis and psittacosis [16] and of trachoma and inclusion conjunctivitis,[17] but all these agents appear to share a common antigenic factor which results in cross reactions in the complement fixation test.

---

15. Grace, A. W.; Shaffer, M. F., and Rake, G.: Further Evidence Concerning the Specificity of the Lymphogranuloma Venereum Complement-Fixation Test in Syphilis, Am. J. Syph., Gonor. & Ven. Dis. **27**:44, 1943.

16. Rake, G.; Eaton, M. D., and Shaffer, M. F.: Similarities and Possible Relationships Among Viruses of Psittacosis, Meningo-Pneumonitis, and Lymphogranuloma Venereum, Proc. Soc. Exper. Biol. & Med. **48**:528, 1941.

17. Rake, G.; Shaffer, M. F., and Thygeson, P.: Relationship of Agents of Trachoma and Inclusion Conjunctivitis to Those of the Lymphogranuloma-Psittacosis Group, Proc. Soc. Exper. Biol. & Med. **49**:545, 1942.

RELATION BETWEEN TITER OF SERUM AND DIMENSIONS OF FREI REACTION

Early in the study it was noted that the serum of a lymphogranulomatous person who yielded a large cutaneous response to the Frei test generally fixed complement in a higher dilution than serum from a person whose cutaneous reaction was small. The existence of such a relation is shown in table 3, in which the data pertaining to 107 persons are presented in 3 groups delimited by an arbitrarily chosen minimum titer of serum. The demonstrated relationship suggests either that the circulating and sessile antibodies are the same or that the concentration of one rises *pari passu* with that of the other.

The ratio of the lowest to the highest average titer of serum—approximately 14—is much greater than the corresponding figure—approximately 2—for the size of the Frei reaction, a fact which should make the complement fixation the more sensitive test of diminution of intensity of infection. The variation in size of cutaneous response among different subjects is less when the serum titer is high than when it is low, and no person in whom the latter was as high as 1 : 240 showed a negative cutaneous reaction. The "maximum" (and "minimum") figures in the two halves of table 3 do not relate in any of the three groups to the same subject. Such relationship between the cutaneous response and the titer of complement-fixing antibodies would be understandable on the basis of the cutaneous reaction's possessing a higher threshold. If to this is added the fact of the well known individual differences in degree of cutaneous response, the results become readily explicable. When a high serum titer is present, the threshold for cutaneous response is passed by so great a margin that even persons whose skin reacts only weakly under all conditions will give a positive reaction. In the presence of low serum titers the threshold for cutaneous response is passed only in persons whose skin reacts strongly under all conditions.

TABLE 3.—*Relation Between Complement-Fixing Titer of Serums of Lymphogranulomatous Persons and Size of Frei Reaction with Yolk Sac Antigen*

Number of Subjects	Titer of Serum			Size of Papule in Frei Reaction, Mm.		
	Maximum	Average	Minimum	Maximum	Average	Minimum
18	1:1920	1:552	1:240	26 by 20	16 by 14	10 by 10
24	1:120	1:90	1:60	25 by 15	10 by 8	3 by 2
65	1:30	1:14	1:6	16 by 16	7 by 6	0

COMPLEMENT-FIXING TITER OF SERUMS FROM PERSONS WITH SYMPTOMATIC AND WITH ASYMPTOMATIC LYMPHOGRANULOMA VENEREUM

The symptomatic entities dealt with in this section are proctitis with stricture and inguinal adenitis, suppurative and nonsuppurative and without anorectal lesions. All subjects showed active clinical manifestations at the time that the serums were taken for testing.

Symptomatic Manifestations.—Proctitis with Stricture: This is the commonest [18] and most specific manifestation of lymphogranuloma venereum, being associated with a positive Frei reaction in 96.8 per cent of cases.[19] The average titer of 34 serums was 1 : 213, with limits of 1 : 1,920 and 1 : 6.

18. Grace, A. W.: Lymphogranuloma Venereum, Bull. New York Acad. Med. **19**:627, 1941.

19. Grace, A. W.: Anorectal Lymphogranuloma Venereum, J. A. M. A. **122**:74 (May 8) 1943.

Inguinal adenitis: The average titer of 12 serums was 1:98, with limits of 1:480 and 1:6.

Asymptomatic Manifestations.—The serums of 32 persons who had neither a history nor clinical manifestations of lymphogranuloma venereum fixed complement at an average titer of 1:32, with limits of 1:120 and 1:6. It might be advanced in explanation of the almost sevenfold increase in the titer of the symptomatic as compared with the asymptomatic disease that the patient with proctitis with stricture, a chronic condition, had had the disease for a longer period than his asymptomatic confrère. Complete comparison of the duration of the disease in the two groups is impossible, as no means exist for determining when the asymptomatic subject acquired his infection. A lead, however, suggesting that a higher titer of serum is not linked entirely with longer duration of the disease is given by the fact that the average age of the persons comprising each group was about the same, which indicates an approximately equal

TABLE 4.—*Complement-Fixing Titer of Lymphogranulomatous Serums After Therapy with Sulfonamide Compounds*

Patient	Amount of Sulfonamide Compound, Gm.	Duration of Therapy, Months	Serum Titer after Therapy
A. M.	730.2	24	1:60
O. S.	693.3	24	1:6
A. E.	683.4	9	1:30
H. K.	620.4	13	1:15
M. P.	424.3	24	1:120
B. R.	391.3	10	1:30
J. F.	338.0	6	1:120
E. T.	321.6	5	1:480
E. C.	264.6	7	1:960
J. H.	255.3	8	1:90
M. T.	193.2	6	1:120
J. R.	186.0	2	1:120
B. S.	142.8	5	1:120
E. C.	126.0	3	1:120
B. G.	118.8	16	1:60
W. C.	94.5	6	1:30
J. D.	91.8	3	1:240
N. M.	84.0	6 weeks	1:240
J. S.	75.0	5 weeks	1:60

period of sexual activity, and, moreover, in 18 persons in whom the disease was acquired between 1914 and 1934 the average titer was 1:204, as compared with 1:168 in 36 infected between 1934 and 1941, a difference without statistical significance. It seems more probable that the most important point is the actual mass of virus antigen to which a person has been exposed. This, in turn, is determined by the extent and duration of an active lesion in which the virus is multiplying.

EFFECT OF SULFONAMIDE COMPOUNDS ON THE COMPLEMENT-FIXING TITER OF LYMPHOGRANULOMATOUS SERUMS

The serums of 19 persons were examined at the end of a period of therapy with sulfanilamide or sulfathiazole or both, and the results are shown in table 4. All had the disease in the anorectal area except J. S., whose bladder was involved. For 2 patients only, C. S. and H. K., was the serum tested before and after the use of the drugs, and in both treatment had no effect on the titer. Serum titers of not less than 1:30 were found in 17 persons after treatment. Whether or not

complement-fixing antibodies can be made to disappear by the use of concentrations of sulfonamide compounds higher than those reported here is a matter for further study.

SUMMARY

Antigens made from lymphogranulomatous nodes or pus are unsatisfactory for the performance of the complement fixation test for lymphogranuloma venereum owing to their low content of virus. The new yolk sac antigens can be prepared with any desired concentration of virus. With them it has been shown that the complement fixation test is more sensitive than the Frei test, that a relation exists between the average titer of lymphogranulomatous serum and the average dimensions of the Frei reaction and that the average titer in certain symptomatic lymphogranulomatous entities is considerably higher than that in asymptomatic lymphogranuloma venereum. Prolonged therapy with sulfonamide compounds in moderate doses did not remove the complement-fixing antibodies from the serum of the lymphogranulomatous persons considered in this study, in whom perhaps the infection was not entirely cured but only altered into an inactive, latent, form.

11 Schermerhorn Street.
The Squibb Institute for Medical Research.

SYPHILITIC JUXTA-ARTICULAR NODULES

FREDERICK KALZ, M.D.
AND
B. L. NEWTON, M.D.
MONTREAL, CANADA

Recently the subject of juxta-articular nodes as a manifestation of syphilis has aroused considerable interest in North America. However, of almost 200 cases reported from nontropical regions, only about 30 have been observed on this continent. According to Hudson,[1] between 1 and 2 per cent of the population between the ages of 30 and 50 in the tropics is affected. The scarcity of reported cases in nontropical regions is, we believe, due in part to incorrect clinical diagnosis, and pertinent reasons will be mentioned later.

The purpose of this paper is to report a typical case and to review briefly the historical aspects of and the American literature on this subject.

Juxta-articular nodes may be defined as painless, slowly growing subcutaneous fibrous nodules, often symmetric in their distribution and if untreated of long duration. They are situated in the vicinity of a joint, especially of the extremities, and are not attached to the skin or related to bursas or tendons. Sites of predilection, as described by Jeanselme,[2] are, in order of frequency, elbows, knees, tibial tubercles, sacrococcygeal area and costal cage.

The first reference in the literature to this disease is that of Bertin (cited by Burnier[3]), who described such nodes in natives of Guadelupe (1778) and of Santo Domingo (1786) affected with yaws and syphilis. Almost a hundred years later, Fowler[4] presented a case in which there was a history of probable syphilitic infection: Barlow,[5] who had previously attributed such nodules to rheumatic disease, was present at the meeting, heard Fowler's presentation and stated that he could no longer assert that these nodules were always of rheumatoid causation. Lutz[6] in a letter from Honolulu in 1891 mentioned similar cases of subcutaneous nodules and conjectured regarding their possible relationship to syphilis. In 1900, McGregor[7] reported similar nodules among natives of New Guinea afflicted with yaws.

From the Section of Dermatology, Subdepartment of Medicine, Dr. D. Sclater Lewis, Physician in Chief, and the Department of Pathology, Dr. T. R. Waugh, Pathologist in Chief, Royal Victoria Hospital.

1. Hudson, E. H.: Juxta-Articular Nodules in Euphrates Arabs, Tr. Roy. Soc. Trop. Med. & Hyg. **28**:511, 1935.

2. Jeanselme, E., and Rist, E.: Précis de pathologie exotique, Paris, Masson & Cie, 1909, p. 734; cited by Goodman.

3. Burnier, R.: Les nodosités juxta-articulaires et leurs rapports avec la syphilis, Presse méd. **41**:995, 1933 (reference to Bertin in this paper).

4. Fowler, J. K.: Subcutaneous Nodules Occurring in an Adult Not the Subject of Rheumatism, Brit. M. J. **1**:107, 1884.

5. Barlow, T., and Warner, F.: On Subcutaneous Nodules Connected with Fibrous Structures, Occurring in Children the Subjects of Rheumatism and Chorea, Tr. Internat. M. Cong. **4**:116, 1881.

6. Lutz, A.: Brief aus Honolulu, Monatsh. f. prakt. Dermat. **13**:488, 1891; cited by Hopkins.[22]

7. McGregor, W.: Some Problems of Tropical Medicine, Lancet **2**:1055, 1900.

It was Jeanselme, however, who first described these lesions fully (1899) and suggested the term "juxta-articular nodes." [8] In 1904-1905 he published further observations [9] with a pathologic description. These he first attributed to yaws, but he later stated that there were no changes characteristic of either this disease or syphilis. In 1924 he published another paper, in which he discussed the histologic changes in detail.[10] In Paris in 1927, one of us (F. K.) had the opportunity of seeing 2 patients presented by Jeanselme, who stated at this time that the Wassermann reactions of all the patients he had seen recently had been positive and that the earlier negative results were probably due to lack of sensitivity of the test.

Since 1927 cases have been reported from tropical and from nontropical regions of nearly every part of the world. In 1933, Hu and Frazier [11] reported in detail several cases from China and presented a survey of the literature up to that time. In the same year, a critical review of this subject, including bacteriologic observations, was published by Burnier.[3] Fenner [12] also reported the incidence of juxta-articular nodes among the aborigines of Australia; his article contains an interesting map showing the geographic distribution of all cases reported up to 1938. In spite of the widespread distribution, up to the present time there have not been any cases reported from the west coast of the United States or from Canada.

Of particular interest are the views of Hudson,[1] who studied the epidemiology of bejel, which is now recognized as syphilis rather than yaws or a disease of separate entity. He found that the incidence of juxta-articular nodes in syphilis was as high as that reported from areas where yaws was present (i. e., in the tropics) if certain epidemiologic conditions were present. Universal prevalence, childhood acquisition and lack of treatment are the determining factors. Both Jeanselme [13] and Jessner,[14] the latter a member of the Russo-German expedition for the study of syphilis in Siberia,[15] stated that there is not any way of differentiating the nodes of syphilis from those of yaws and that the histologic pictures may be identical; this would be used as an argument in favor of the unitarian concept of these two diseases.

The first case reported in North America was that of Goodman and Young [16] in 1921. These authors recognized the similarity in many respects of their case to those reported by Jeanselme. Two further papers on this subject were later

8. Jeanselme, E.: La syphilis dans la péninsule Indo-Chinoise, Ann. de dermat. et syph. **2**:817, 1901.

9. Jeanselme, E.: Nodosités juxta-articulaires, Compt. rend. Soc. de méd. et d'hyg. coloniales, 1904, p. 15; cited by Gutierrez [20]; Des nodosités juxta-articulaires observés sur indigènes de la Presquile Indo-Chinoise, Rev. de méd. et d'hyg. trop. **2**:11, 1905.

10. Jeanselme, E.: Juxta-articulaires nodosités, Paris méd. **1**:242, 1924; cited by Greenbaum.[19]

11. Hu, C. K., and Frazier, C. N.: A Study of Subcutaneous Nodules of the Juxta-Articular Type Observed in Five Cases of Syphilis in North China, Chinese M. J. **47**:364 1933.

12. Fenner, F. J.: The Occurrence of Juxta-Articular Nodules in Australia, M. J. Australia **2**:412, 1938.

13. Jeanselme, E.: Sur la structure des nodosités juxta-articulaires, Bull. Soc. path. exot. **9**:287, 1916; cited by Fox.[18]

14. Jessner, M.: Ueber juxta-artikuläre Knotenbildungen, Klin. Wchnschr. **3**:1499, 1924.

15. Jessner, M., and Rossiansky, N.: Die Ergebnisse der deutsch-russischen Syphilis-Expedition 1928, Arch. f. Dermat. u. Syph. **160**:224, 1930.

16. Goodman, H., and Young, W. J.: A Clinical Pathological Study of an Unusual Syphilitic Manifestation Resembling Juxta-Articular Nodules, Am. J. M. Sc. **159**:231, 1920.

published by Goodman.[17] In 1922, Fox [18] described lesions occurring in a Negro, which were in many ways similar to those in our patient. These papers aroused considerable interest, and publications by Greenbaum,[19] Gutierrez,[20] Brunsting,[21] Hopkins,[22] Greenbaum and Cobane,[23] Sulzberger [24] and Zakon and Allan [25] followed in the order mentioned from 1924 to 1940. Freeman [26] and Tuta and Coombs [27] reported cases of juxta-articular nodes in 1941 and 1942, respectively.

The few cases reported seem to indicate that this condition is comparatively rare in North America, but presentation of a patient with such nodes at a medical meeting has always evoked comments regarding the similarity of the lesions to some previously observed. This undoubtedly indicates that many such lesions have been diagnosed as fibroma in the past. We agree with Burnier [3] that the concept of a syndrome of multiple causes has much to support it, for similar nodes may also occur in rheumatic disease, filariasis, acrodermatitis chronica atrophicans, scleroderma, calcinosis, gout and xanthoma. Differentiation between the nodes of syphilis and those of rheumatism may present difficulty, since both are common diseases and may occur in the same person.

In some instances, no suspicion as to the syphilitic origin has arisen before histologic examination of tissue from the node. Milch and Galland [28] collected several cases of tumor-like lesions and emphasized the tendency of Treponema pallidum to produce fibrous masses in and about the muscles. The observations in one of his cases fit readily into the group of syphilitic juxta-articular nodules. Tuta and Coombs [27] in a recent report admitted that syphilis was not considered and that it was only after histologic examination that the correct diagnosis was made. However, histologic features can be deceptive. Freeman [26] has reported cases in which the appearances were those of hyaline fibrous tissue without cellular infiltration. Such reports emphasize the inadequacy of any single method in diagnosing this disease. The presence of syphilis, diagnosed clinically and serologically, a carefully taken history, a histologic examination and observation of the subsequent course are all essential in order to diagnose this disease correctly.

These nodules have been referred to by different authors as juxta-articular nodosities, multiple juxta-articular fibrogummatous nodules, subcutaneous fibroid syphilomas, tumor fibrosis syphiliticus, juxta-articular syphilitic nodosities, para-articular nodosities and subcutaneous nodular syphilomas. We feel that Jean-

17. Goodman, H.: Chronic Fibroid Subcutaneous Syphilomata, Brit. J. Dermat. **33**:335, 1921; Fibroid Subcutaneous Syphilomata, Am. J. Syph. **6**:687, 1922.

18. Fox, H.: Subcutaneous Fibroid Syphilomas of Elbows and Knees, Arch. Dermat. & Syph. **5**:198 (Feb.) 1922.

19. Greenbaum, S. S.: Fibroid Gumma, J. Lab. & Clin. Med. **10**:476, 1925.

20. Gutierrez, P. D.: Nodosités Juxta-Articulaires, Arch. Dermat. & Syph. **12**:159 (Aug.) 1925.

21. Brunsting, L. A.: Syphilitic Juxta-Articular Nodes, Am. J. Syph. **15**:42, 1931.

22. Hopkins, H. H.: Subcutaneous Nodules of the Juxta-Articular Type, Bull. Johns Hopkins Hosp. **49**:5, 1931.

23. Greenbaum, S. S., and Cobane, J.: Syphilitic Fibrosis and the Status of the Iodides in the Present Day Treatment of Syphilis, Am. J. Syph. **18**:289, 1934.

24. Sulzberger, M. B.: Juxta-Articular Nodules in Syphilis, Arch. Dermat. & Syph. **34**: 337 (Aug.) 1936.

25. Zakon, S. J., and Allan, H. S.: Palmar Juxta-Articular Nodes, Quart. Bull., Northwestern Univ. M. School **14**:270, 1940.

26. Freeman, H. E.: Juxta-Articular Nodules, Tertiary Syphilis with Aortic Insufficiency and Dermatitis Due to Mercupurin, Arch. Dermat. & Syph. **43**:206 (Jan.) 1941.

27. Tuta, J. A., and Coombs, R. A.: Symmetric Syphilitic Granulomas of the Elbow, Arch. Dermat. & Syph. **46**:375 (Sept.) 1942.

28. Milch, H., and Galland, W.: Pseudoneoplastic Luetic Granulomata, Am. J. M. Sc. **182**:405, 1931.

selme's term, juxta-articular nodules, is best suited to describe this disease because it is internationally accepted by the majority of authors, is descriptive and permits the inclusion of the etiologic factors.

REPORT OF A CASE

C. D., a 71 year old Negro, was admitted to the Royal Victoria Hospital because of pains in the right ankle. He was born in the United States but had lived most of his life in Canada, where he was employed as a railway worker and later as a janitor. He had never left the North American continent. He gave a history of gonorrhea several times and a painful ulcer on the penis in 1907, which he treated with cupric sulfate. Blood tests had never been made, and he was unaware of his syphilitic infection. He stated that one year after the appearance of the penile lesion he noticed a small hard growth on the right

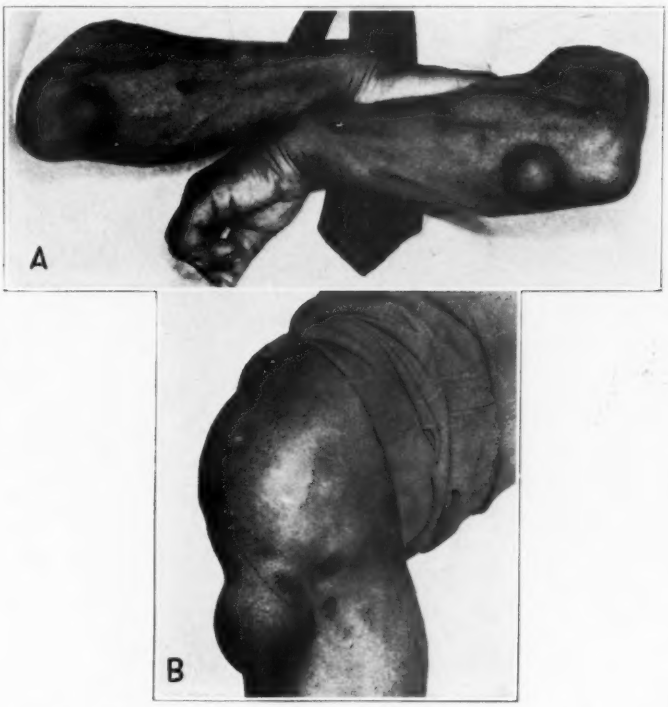


Fig. 1.—*A*, juxta-articular nodule of thirty years' duration on the left elbow, and one of fourteen years' duration on the right elbow of a syphilitic Negro. *B*, juxta-articular nodule of thirty-five years' duration on the left knee.

knee, and shortly afterward a similar node appeared on the left knee; this disappeared spontaneously after a fall on that knee. During this period his work involved kneeling. About 1912 he noticed a similar node on the left elbow, and in 1928, twenty-one years after the appearance of the first growth on the right knee, a small tumor appeared on the right elbow. These tumors grew slowly for many years without causing any discomfort until shortly before he was first seen at the hospital, at which time he had no complaints other than the pain in his right ankle and some shortness of breath, which he attributed to his age.

The family history revealed that he had a daughter, aged 22, alive and well and that his wife had died many years ago of unknown cause; she had never had a miscarriage or stillbirth.

Physical examination showed the patient to be a well nourished Negro, looking much younger than his years and in apparently good health. He presented no stigmas of congenital syphilis. The pupils were unequal, the right being slightly irregular with sluggish reaction to light. A complete neurologic examination revealed no further abnormalities; there were

no decided mental changes, although the patient appeared rather euphoric and forgetful. The heart and aorta were greatly enlarged on percussion, and an aortic diastolic murmur together with venous pulsations on the right side of the neck were present. His blood pressure was 170 systolic and 90 diastolic in both arms; thrills were not felt, and no signs of decompensation were present. The liver was not enlarged. A large scar was seen on the dorsum of the penis. There were hard painless nodules the size of a chestnut on both forearms along the ulnar borders and a similar, somewhat larger, round tumor on the left knee.

The nodes were not lobulated and were not sensitive to pressure; they were hard but by no means cartilaginous, more of a rubbery consistency. There was no fluctuation, and the overlying skin was normal in appearance and freely movable over the nodules, which were partially movable.

On the right external malleolus tumors the size of small beans were palpated. These nodules, not visible on inspection, were revealed by roentgen examination and reported on as follows: "The soft tissue about the right heel, on both the posterior and the plantar

Fig. 2.—Section of a nodule from the left knee. This section demonstrates the central area of degenerative change within fibrous-like tissue.

surface, was thickened and somewhat of the same character as the masses about the left knee and the elbows."

The Wassermann reaction of the blood was positive with a dilution of 1:256; the Kahn reaction was positive, and examination of the cerebrospinal fluid revealed the Wassermann reaction to be positive in 1 cc., doubtful in 0.6 cc. and negative in 0.4 cc. The colloidal gold curve was normal; the total protein content was 33 mg. per hundred cubic centimeters; the Pandy reaction was negative, and there was no increase in cell count.

Roentgen examination of the chest revealed that the heart was enlarged to the left, and a diffuse widening of the aorta was noted involving chiefly the arch and the descending portion. Some calcification of the wall had occurred, and the right innominate artery was widened and pulsating, suggesting an aneurysm. The esophagus was not displaced, but barium sulfate was retained up to the dilatation of the aorta. The pulmonary fields were clear, and the diaphragm moved freely.

A diagnosis of late syphilis with juxta-articular nodes, aortitis and aneurysm of the innominate artery and asymptomatic inactive neurosyphilis was made.

The nodules were surgically removed for histologic examination. The operation did not present any difficulties and the nodes were easily removable, but the wounds healed somewhat slowly.

Antisyphilitic treatment with bismuth subsalicylate and potassium iodide was started.

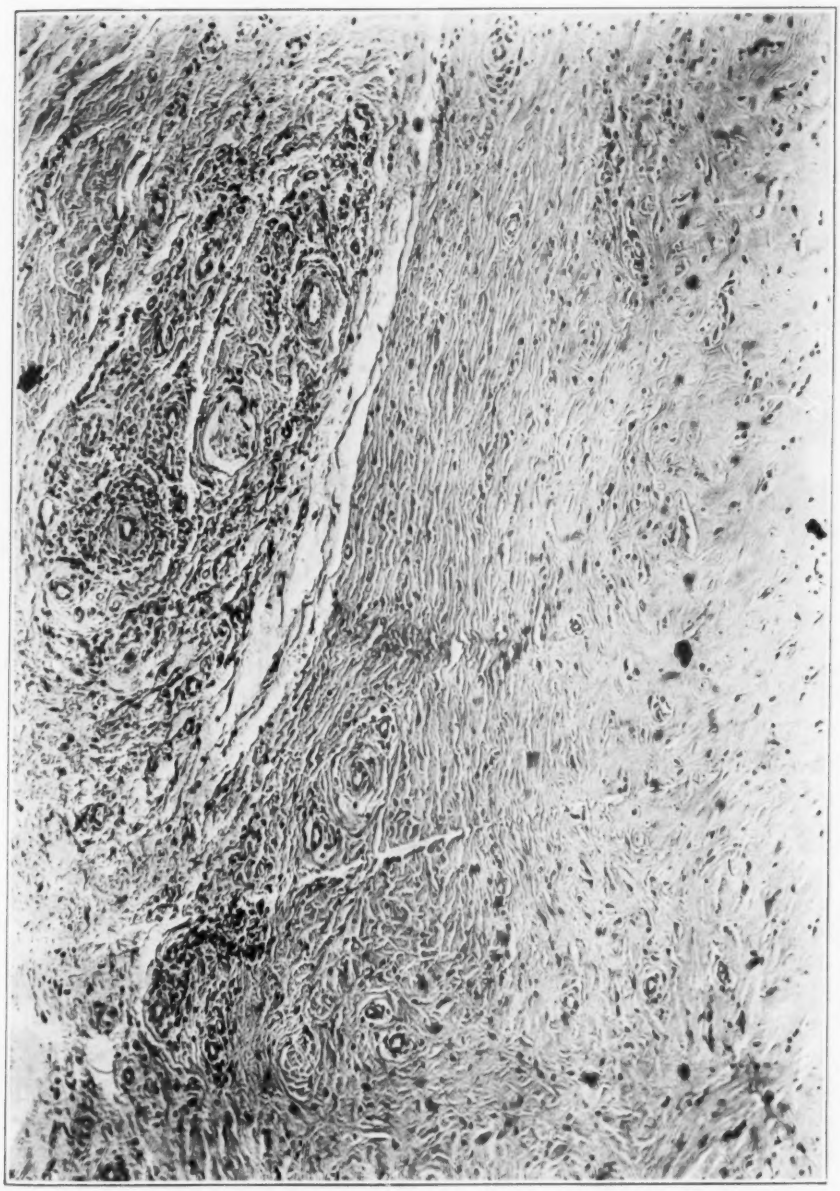


Fig. 3.—Section of nodule from left knee. This illustrates the peripheral perivascular inflammatory changes and the increasing density of the fibrous tissue toward the middle of the nodule (hematoxylin and eosin, Masson's trichrome and Van Gieson stains; × 80).

Gross pathologic examinations of the operative specimens showed three round to oval grayish pink rubbery masses.

The largest nodule was taken from the knee and measured 8 by 6 by 7 cm., and the two smaller ones, from the elbows, measured 5 by 3.5 by 4 and 4.5 by 2.5 by 2.5 cm. respectively.

On section the material from the largest lesion presented a firm wall approximately 1 cm. in thickness within which was a network of fiber-like whitish to yellowish green tissue. Between the fibers of this network were thin clefts and cystic spaces containing greenish fluid. The two smaller masses were firmer than the large one and on section presented a more uniform appearance, with whitish firmly cut surfaces somewhat suggestive of fibroma. Only a few small cystlike spaces were seen, and a correspondingly small amount of greenish fluid, similar to that found in the larger tumor, was present.

Microscopic examination of the material presented a rather unusual picture (fig. 3).

Toward the external aspect, the tissues were more loosely arranged, with several small blood vessels scattered throughout. These showed thickening of walls and were surrounded by lymphocytes, plasma cells and a few polymorphonuclear leukocytes. Loss of structural detail was present, and less cellular exudate was seen as the center of the mass was approached, with thick hyaline fibrous septums ramifying to form partitions for cystic spaces. These spaces contained structureless masses of material staining pink with eosin. Here and there clefts probably due to dissolved cholesterol crystals were visible. Discretely scattered among the debris were small foci of lymphocytes, plasma cells and polymorphonuclear leukocytes. In a few small areas, the histologic structure was slightly suggestive of gummatous necrosis, and in one of these a single atypical giant cell was seen, but no tendency to form typical tubercles was present.

The two smaller tumors, of more recent origin, presented a more fibromatous appearance throughout, consisting of whorls and bundles of dense fibrous tissue and collagen fibers. Hyalinization and loss of structural detail, although present, were considerably less than in the material from the knee, and only a minimal amount of cystic degenerative changes was seen. The type of cellular exudate here was essentially the same as that previously described.

Although Aravijskij and Bulvachter[29] reported that Treponema pallidum had been found in a nodule of a patient with syphilis, all our attempts to demonstrate the presence of microorganisms yielded negative results.

Jessner[30] and others reported the inoculation of a portion of a nodule into rabbits, in which syphilis subsequently developed, but as our material was received fixed in 40 per cent solution of formaldehyde no attempt at animal inoculation was made.

COMMENT

The onset of the syphilitic infection in this patient could not be established with certainty. The fact that the first node appeared one year after the penile ulcer may be significant, as cases have been reported in which nodes occurred as early as three months after the infection.[3] In this respect, a close parallel may be seen between our case and the one described by Fowler.[4] In the majority of cases, however, the nodes have appeared many years after the infection.

The racial background of the patient may be of interest, in view of the tendency to formation of exuberant fibrous tissue in Negroes. Goodman[16] and Fox[18] both mentioned a possible connection in this respect; however, in many of the cases reported from the temperate zone the nodes occurred in persons of the white race. The occupation of our patient at the time the nodes first appeared was one which forced him to work on his knees; Jeanselme,[31] Sweitzer and Winer[32] and others considered that trauma may possibly be a precipitating factor, and Sweitzer and Winer[32] published the photograph of the lesion and reported the histologic observations in a case in which oft repeated slight trauma was the sole etiologic factor in a nonsyphilitic patient with juxta-articular nodes.

---

29. Aravijskij, A., and Bulvachter, A.: Zur Frage der symbiotischen Infektion bei syphilitischen juxta-artikularen Verhartungen, abstracted, Zentralbl. f. Haut- u. Geschlechtskr. **39**: 443, 1931-1932.

30. Jessner, M.: Ueber syphilitische juxta-artikuläre Knotenbildungen, Arch. f. Dermat. u. Syph. **152**:132, 1926; cited by Brunsting.[21]

31. Janselme, E., and Schulmann, E.: Traité de la syphilis, Paris, Gaston Doin, 1932, vol. 3; cited by Burnier.[3]

32. Sweitzer, S. E., and Winer, L. H.: Fibrotic Nodules of the Skin, Arch. Dermat. & Syph. **45**:315 (Feb.) 1942.

The long asymptomatic course of the nodes is a common observation, and the presence of other late syphilitic manifestations has often been reported. In this respect our case is similar to that described by Fox.[18]

The roentgen observations of the lower extremities are of importance. As previously stated, we missed the nodes which were not visible on clinical examination. Such small though palpable nodules may occur frequently and most likely escape the attention of the examiner in a routine case of syphilis.

With regard to pathologic changes in our case, the three zones of Jeanselme [13] were clearly seen in the nodule from the knee. In brief, the changes he described were an outer or peripheral zone of mononuclear cell exudate, together with dilated blood channels in a loose connective tissue; an intermediate fibroma-like scar or tissue zone; and an inner zone of hyalinized tissue, with amorphous material. The cystlike degeneration, most evident in the large nodule, is a rare finding and has not been previously reported in the literature from North America. In our opinion, these changes are probably related to the extremely long duration of the nodules, at least in some cases. The smaller nodes showed a fibroma-like structure throughout and belong to the type of nodule which might be easily mistaken for fibroma by an unsuspecting surgeon or pathologist.

Nothing of further interest was observed in our case other than the decrease in size of the small nodules on the right ankle after three months' treatment with bismuth subsalicylate.

Visualizing the whole course in the light of observations on our patient and of other observations reported in the literature, one may assume that in all probability a nodule first arises as a small granuloma recognized only by palpation; this may regress spontaneously or grow. If it is detected while still in the granuloma stage, a rapid response to therapy may be observed; if it is untreated, slow continuous growth occurs with fibroplastic increase, giving rise to tumor-like characteristics in many instances. Antisyphilitic therapy given during this stage may be successful, but little or no response to treatment is to be expected in long-standing lesions if complete fibrosis with hyalinization occurs. Manifestations of degeneration may appear, especially in nodes of large size. Some authors [33] have agreed that response to antisyphilitic therapy is rapid and complete, while Weber [34] and Fox [18] described a slower or only partial effect from such therapy. These contradictory reports on the effect of antisyphilitic therapy may be explained by the various stages of this condition when treatment was instituted.

One of us (F. K.) had the opportunity of seeing similar nodules in a 42 year old Polish woman unaware of her syphilitic infection. She had a hard cherry-sized tumor close to the right elbow (permission for excision could not be obtained) and a circinate nodular syphilid on the extensor surface of the right arm. Histologic examination of tissue from this syphilid revealed foci of tuberculoid structure almost indistinguishable from lupus vulgaris. This syphilid disappeared after three weeks of therapy, and the node gradually decreased about 50 per cent in size during the ensuing two years of intensive antisyphilitic treatment.

SUMMARY

A brief historical review of the literature demonstrates that the clinical entity and syphilitic origin of juxta-articular nodes were described more than one hundred and fifty years ago.

33. Burnier.[3] Sulzberger.[24] Goodman and Young.[16] Goodman.[17]

34. Weber, F. P.: Chronic Fibroid Subcutaneous Syphilomata of the Legs, Associated with Chronic Peri-Urethral Induration in the Penis (So-Called "Induratio Penis Plastica"), Brit. J. Dermat. **32**:173, 1920.

The majority of cases have been observed in the tropics or in regions where syphilis or yaws has an endemic character.

There is some evidence that in many cases such nodes have been incorrectly diagnosed owing to the fact that neither the clinical nor the histologic picture alone is an adequate criterion for diagnosis.

In a case of juxta-articular nodes of many years' duration occurring in an American Negro, the histologic examination demonstrated extensive degenerative changes in some nodes, in which the three zones described by Jeanselme were observed, while the more recent nodes resembled simple fibroma.

Dr. Gavin Miller, department of surgery, Royal Victoria Hospital, referred this patient to us. Dr. Carleton Peirce, department of radiology, and Dr. T. R. Waugh, department of pathology, Royal Victoria Hospital; Dr. G. L. Duff, department of pathology, McGill University, and Dr. J. E. Pritchard, department of Pathology, Montreal General Hospital, cooperated in the study of the case.

VIRUS DISEASES OF SKIN, WITH SPECIAL REFERENCE TO ELEMENTARY AND INCLUSION BODIES

I. VARIOLA-VACCINIA AND MOLLUSCUM CONTAGIOSUM

MICHAEL H. EBERT, M.D.
AND
M. OTSUKA, M.D.
CHICAGO

Virus diseases of plants and animals have always played an important role in human economy. The earliest triumphs of preventive medicine were achieved in this field before the nature of such diseases was understood. Their extraordinary capacity to flame up into world-wide epidemics makes their study of primary interest during a world war. The dermatologist has an excellent right to a special interest in virus diseases. The first successful attempt to produce artificial immunity was in smallpox. Most of the infectious exanthems are of virus or rickettsial origin. Pioneer work by Guarnieri, Paschen, von Prowazek, Lipschütz and others on the histology of virus diseases was made possible by the easy accessibility of cutaneous lesions. The intensive study of the cytologic changes caused by the viruses of herpes simplex, herpes zoster, molluscum contagiosum, varicella, variola and others by outstanding students of virus diseases testifies to the opportunity offered by diseases of the skin. It is also a matter of patriotic pride to know that the outstanding contributions to the study of viruses in the last twenty-five years have been made by English-speaking investigators.

It would be out of place here to discuss the nature of the virus. Stanley's discovery that the virus of tobacco mosaic is a crystalline protein no larger than several other organic molecules has made such discussion difficult. It is natural to compare the viruses which cause disease with bacteria, which are longer known and better understood. Viruses are smaller than bacteria. However, their distinguishing characteristics are chemical rather than physical. They differ fundamentally from bacteria in that they are unable to carry on biologic oxidation. Hence, they can multiply only in living cells and cannot be cultivated on artificial mediums. Rickettsias do not differ fundamentally from viruses except that they are transmitted by an arthropod vector.

Our interest has centered chiefly on the demonstration and significance of elementary bodies and inclusion bodies in virus diseases of the skin. While no claim is made to originality, it has seemed worth while to rework the field and assemble photomicrographs of our preparations, inasmuch as the original material is widely scattered throughout the literature.

Two apparently unrelated diseases, the variola-vaccinia group and molluscum contagiosum, have been chosen for this preliminary paper, for a variety of reasons: Both viruses are large and consequently relatively easy to demonstrate. Thus, they make excellent test objects for microscopic study, particularly the virus of molluscum contagiosum, which is easy to obtain and is the largest virus affecting the human skin. The inclusion bodies of these two diseases were the first to be described, although their real nature was not originally recognized. The elementary

From the Dermatological Department, Cook County Hospital.

bodies of variola-vaccinia were the first to be stained and demonstrated microscopically. Because of the epidemiologic importance of variola, this virus has probably been more intensively studied than any other. Finally, variola-vaccinia and molluscum contagiosum offer an excellent study in contrasts. One is a systemic infection and the other a purely local one. Consequently, the immune processes are strikingly different.

For the purposes of this paper the viruses of variola and vaccinia have been treated as identical. It is still a controversial question whether vaccinia virus is a biologic mutant of variola virus or a closely related virus derived from a parent strain. The elementary bodies are morphologically identical and the inclusion bodies closely similar.

THE ELEMENTARY BODY

The study of virus diseases has been handicapped not only by the impossibility of cultivating the virus on nonliving mediums but by the difficulty in demonstrating them microscopically. Even the largest viruses cannot be visualized with ordinary stains. With special staining methods minute bodies can be demonstrated in preparations made from virus-infected tissue. These are called elementary bodies.

THE ELEMENTARY BODIES OF VARIOLA-VACCINIA

Buist,[1] of Edinburgh, in 1886 stained smears from vaccinia pustules with aniline methyl violet. He described minute spherical bodies in these smears and estimated their diameter to be 0.150 micron. He believed they were the causal organism of vaccinia. He was a man far ahead of his time. His contemporaries were not merely skeptical; they were not even interested. In 1904 Borrel [2] stained smears from fowlpox, an infectious epitheliosis of chickens which bears many resemblances to molluscum contagiosum. He used Loeffler's flagella mordant of iron tannate followed by Ziehl's aniline fuchsin and described numerous minute spherical bodies. These bodies in fowlpox have subsequently been termed Borrel bodies. In 1906 Paschen [3] took smears from a vaccination vesicle, fixed them in absolute alcohol and stained them with alkalinized Giemsa stain. He described numerous minute spherical bodies. In his first paper he merely registered the facts, making no claims for their etiologic significance. He also observed with dark field illumination numerous minute bodies in fresh lymph from a vaccination vesicle. He then adopted essentially the same stain used by Borrel for fowlpox, i. e., Loeffler's flagella mordant followed by Ziehl's carbolfuchsin, which since has gone by his name and is known as Paschen's stain. In short order he [4] demonstrated the same minute bodies in smears from a variola pustule, vaccinia inoculation on rabbit's cornea and sheep-pox. He now considered the elementary bodies identical with the virus and the causal organism of variola-vaccinia. They are now known as Paschen bodies. Mackie and van Rooyen,[5] who recently revived and confirmed Buist's investigations, have suggested that they should be known as Buist-Paschen bodies.

METHODS OF DEMONSTRATION OF ELEMENTARY BODIES

Various methods have been used to demonstrate the elementary bodies of variola-vaccinia, as well as many other viruses. Since the methods used for variola-

1. Buist, J. B.: Proc. Roy. Soc. Edinburgh **13**:603, 1886.
2. Borrel, A.: Compt. rend. Soc. de biol. **57**:642, 1904.
3. Paschen, E.: München. med. Wchnschr. **53**:2391, 1906.
4. Paschen, E., in Kolle, W.; Kraus, R., and Uhlenhuth, P.: Handbuch der pathogenen Microorganismen, Berlin, Gustav Fischer, 1930, vol. 8, p. 857.
5. Mackie, T. J., and van Rooyen, C. E.: Edinburgh M. J. **44**:72, 1937.

vaccinia are the same as those used for other viruses, a brief discussion will be in order.

A. Staining Methods.—I. Paschen's stain: Since this was the method we were compelled to adopt, a detailed description is appended. Owing to the exigencies of war, victoria blue and primulin were not obtainable. Also we felt it was best to master one method before attempting others.

All glassware must be new and scrupulously cleaned with soap and water, chromic acid and distilled water and stored in 95 per cent alcohol. Before a smear is made the slides are flamed and cooled. Smears must be as thin as possible.

Preparation of the stain. Two solutions are required:

1. Loeffler's flagella mordant

20 per cent aqueous solution of tannic acid	100 cc.
saturated aqueous solution of ferrous sulfate	50 cc.
saturated alcoholic solution of basic fuchsin	10 cc.

2. A stain best made up in two parts

Solution *a:*	basic fuchsin	0.3 Gm.
	ethyl alcohol	10.0 cc.
Solution *b:*	phenol	5 Gm.
	distilled water	95 cc.

Solutions *a* and *b* are mixed before use in the proportion of 1 part of *a* and 9 parts of *b*.

1. Make a thin film on a clean slide.
2. Place in distilled water for three minutes.
3. Dry in air.
4. Fix in chemically pure methyl alcohol for three minutes.
5. Dry in air.
6. Cover with Loeffler's mordant; warm until steam arises, and allow it to remain for ten minutes.
7. Wash thoroughly in distilled water.
8. Cover with freshly mixed carbolfuchsin stain; heat gently till steam rises one minute and allow it to remain ten minutes.
9. Wash rapidly in distilled water and dry with filter paper.
10. Decolorize in absolute alcohol if overstained.
11. Mount in cedar oil and cover with cover slip.
    Elementary bodies appear deep red.

Considerable experimentation is required to determine the duration of steaming of the mordant and the carbolfuchsin as well as the amount of decolorization. The stained specimen may be examined with ordinary lighting or with the dark field illuminator. We have found the dark field method far superior for our purposes, especially in the taking of photomicrographs.

Since the elementary bodies are so small, pitfalls are numerous, particularly in the study of the smaller viruses. Common sources of error are:

1. Protein particles. In dried smears the protein apparently coagulates in small aggregates of the same order of size as the virus. When viewed in stained specimens under the microscope, they look like pale shadows of the elementary bodies. They vary in size, however, and never take the stain completely. Every examination for viruses must be preceded by the examination of control smears, similarly stained, taken from vesicles of dermatitis venenata, toxic eruptions, etc., to avoid the error of mistaking protein particles for viruses.

2. Granules from crushed leukocytes stain well but are larger, grouped, less numerous and easily distinguished.

3. Bacteria are considerably larger than elementary bodies. Control smears were stained with Gram stain in every instance. Virus elementary bodies do not take the Gram stains, while bacteria do. The smallest streptococcus or staphylococcus is at least twice as large as an elementary body of molluscum contagiosum or of variola.

Elementary bodies should be present in large numbers, should be all of the same size and should have the same staining properties to indicate a positive result. It must be emphasized that there is no specific stain which will distinguish elementary bodies.

With all these precautions in mind we have prepared excellent specimens from (*a*) a smallpox pustule of four days' duration; (*b*) a rabbit's cornea inoculated with vaccinia virus and removed seventy-two hours later (the technic will be described later under inclusion

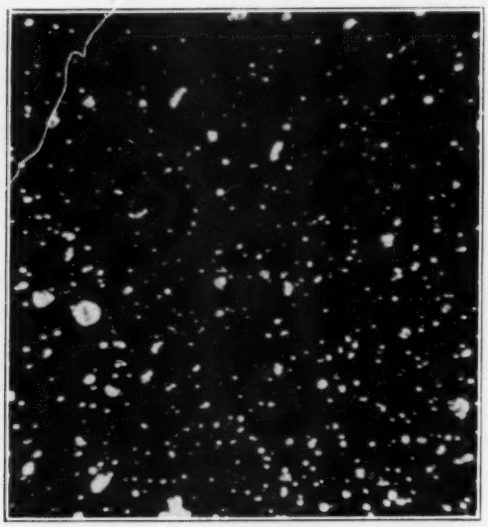


Fig. 1.—Elementary bodies of variola. Smear of the contents of a smallpox pustule taken on the sixth day and stained with Paschen's stain. Dark field illumination and magnification are the same as in figure 4. This was from the first case discovered in the Argo, Ill., epidemic of 1942. There is more surface debris, but the elementary bodies (Paschen bodies) stain well.

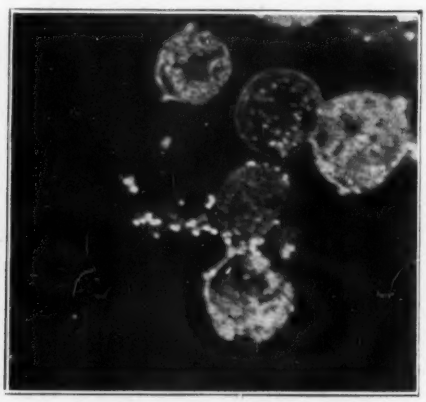


Fig. 2.—Staphylococci and leukocytes stained by Gram's method. Illumination and magnification the same as in figures 1, 4 and 5.

bodies), and (*c*) smears made on the fourth day after the appearance of a vaccinia pustule in a previously unvaccinated person.

Other experimenters have demonstrated Paschen bodies in (*a*) smears from the skin of animals inoculated with vaccinia and cowpox; (*b*) smears as well as fixed preparations from tissue cultures inoculated with vaccinia; (*c*) smears as well as fixed preparations from rabbit testicular tissue inoculated with vaccinia, and (*d*) preparations from the chorioallantoic membrane of chicken or duck eggs inoculated with vaccinia or cowpox. This method,

introduced by Goodpasture[6] in 1931, has proved to be one of the greatest technical advances in virus investigation. Fertile eggs are incubated at 37 to 39 C. for twelve days. With sterile precautions, a triangular window is made over the chorioallantoic membrane, which is exposed and inoculated. The opening is sealed with a sterile cover slip, and the egg is replaced in the incubator for three or four days. Elementary bodies are readily demon-

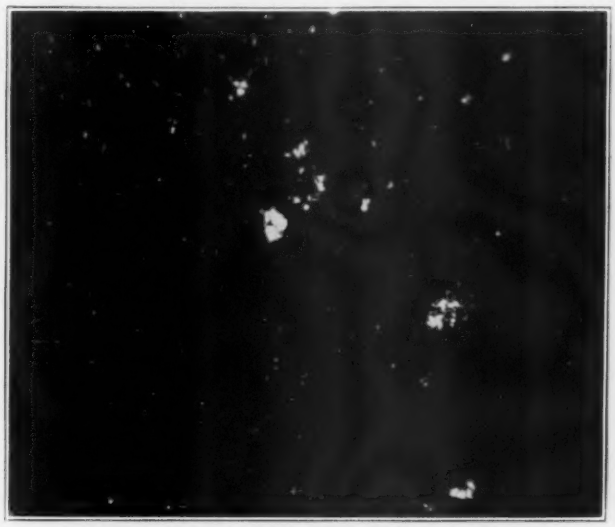


Fig. 3.—Control. Smear of contents of a vesicle of poison ivy dermatitis. Individual protein particles might be impossible to distinguish from elementary bodies in a photograph. However, they vary in size, are not so numerous and do not stain as distinctly. This emphasizes the necessity of concentrating the elementary bodies and separating them from protein by differential centrifugation whenever possible.

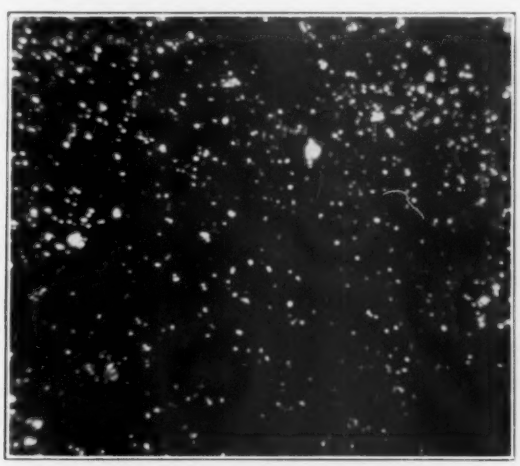


Fig. 4.—Smear made with a droplet of fluid from a vaccination vesicle on the arm of a nonimmune man. Taken three days after the appearance of the first reaction. There are an enormous number of elementary bodies (Paschen bodies) stained with Paschen's stain. Photographed under oil immersion with dark field illumination, the elementary bodies are not all in exactly the same place. The consequent halo on such minute bodies makes some appear larger than others in a photograph. On ocular examination, they all appear the same size.

6. Goodpasture, E. W.; Woodruff, A. M., and Buddingh, G. J.: Science **74**:371, 1931.

strated by direct examination of the living chorioallantoic membrane by annular oblique incident illumination with the Leitz ultropak, as used by Himmelweit,[7] as well as in smears and histologic preparations made from the infected pocks.

II. Victoria blue stain. This was introduced by von Prowazek[8] in 1905, and Herzberg[9] found it more satisfactory than Paschen's stain. Unfortunately we were unable to secure the highly concentrated victoria blue 4 R as prepared by Bayer in Leipzig. Other products proved unsatisfactory. Elementary bodies have been demonstrated by Haagen and Kodama,[10] who used the victoria blue stain on smears taken from the internal organs, i. e., liver, spleen, lungs, kidneys, adrenals and testes, of animals successfully inoculated with vaccinia virus.

III. Morosow's[11] stain is really a silver precipitation method. We have found this an excellent method and simpler than Paschen's stain.

IV. Giemsa stain of good quality properly prepared and buffered gives good results with large viruses. Coles's[12] method is said to be one of the best.

V. Various other methods of staining elementary bodies have been suggested, such as Castenada's,[13] Goodpasture's[14] and Craigie's.[15]

B. Dark field illumination increased the limit of visibility and resolution with small objects. We have already stated that dark field illumination is more satisfactory than direct illumina-

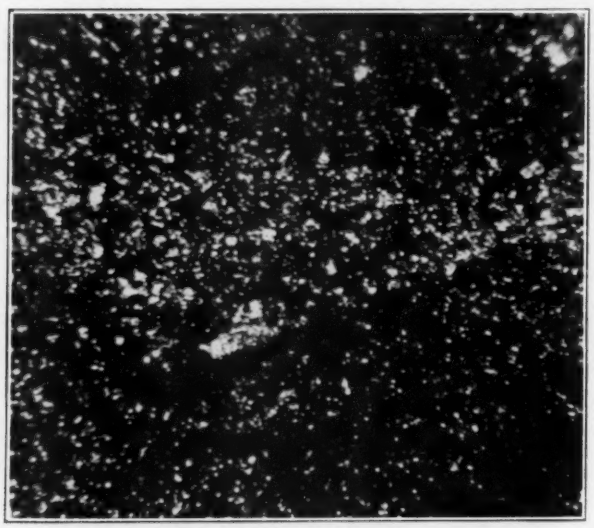


Fig. 5.—Same as figure 4. Taken from a denser portion of the smear. With Gram's stain none of these elementary bodies is visualized.

tion for identifying stained specimens as well as for photomicrography. Elementary bodies can easily be seen in unstained dried smears from rabbits' cornea as well as in saline suspensions. Eisenberg-Merling[16] inoculated rabbits' corneas with vaccinia. On the second and third day the rabbits were killed and a thin shaving of the cornea mounted in isotonic solution of three chlorides (Ringer's solution). With dark field illumination he found elementary bodies in the Guarnieri bodies, free in the cytoplasm of the cells and in the fluid outside the

7. Himmelweit, F.: Brit. J. Exper. Path. **19**:108, 1938.
8. von Prowazek, S.: Arb. a. d. k. Gsndhtsamte. **22**:535, 1905.
9. Herzberg, A.: Zentralbl. f. Bakt. (Abt. 1) **131**:358, 1934.
10. Haagen, E., and Kodama, M.: Zentralbl. f. Bakt. (Abt. 1) **133**:23, 1934.
11. Morosow, M. A.: Zentralbl. f. Bakt. (Abt. 1) **100**:385, 1926.
12. Coles, A. C.: J. Roy. Micr. Soc. **55**:249, 1935.
13. Castenada, M. R.: J. Infect. Dis. **47**:416, 1930.
14. Goodpasture, E. W.: Am. J. Path. **1**:547, 1925.
15. Craigie, J.: J. Path. & Bact. **30**:185, 1933.
16. Eisenberg-Merling, K. B.: J. Path. & Bact. **50**:279, 1940.

cells. Ordinary smears made from the rabbit's cornea seventy-two hours after inoculation with vaccinia virus show the elementary bodies well when examined dry and unstained with dark field illumination.

C. Annular oblique incident illumination with the Leitz ultropak was used by Himmelweit [17] to study living chorioallantoic membranes.

D. Fluorescent microscopy. In the fluorescent microscope the source of light is a mercury vapor lamp emitting rays of a frequency of 2,200 angstroms. These are filtered with a Wood filter, so that the visible rays are retained and the field looks dark. The rays are reflected on a metal mirror through a quartz condenser. The rays which strike particles stained with a fluorescent dye are transformed to rays of a lower frequency in the visible spectrum which can be examined with the ordinary optical system of the microscope. Haitinger [17] did the pioneer work with the fluorescent microscope and suggested its use in the study of viruses and bacteria. Hagemann [18] followed this suggestion and found primulin to be the most satisfactory fluorescent stain for the purpose. Through the courtesy of Dr. Hans Popper, of Cook County Hospital, we made preliminary studies. Elementary bodies stained with primulin look like minute points of light on a dark background. We were unable to secure a sufficient supply of primulin to continue this phase of the work.

E. Ultraviolet rays have been used as a source of illumination to increase the limit of resolution of small objects, because optically the resolving power of a microscopic objective is directly proportional to the wavelength of light employed. Barnard.[19] using a wavelength of 0.255 micron, which is half that of ordinary light, was able to double the limit of resolution of stained viruses. This necessitates the use of a quartz optical system and while ingenious offers no great advantages.

F. The electron microscope has been successfully used to study the detailed structure of the elementary body of vaccinia by Borries, Ruska and Ruska.[20] Recently Green, Anderson and Smadel [21] have presented photographs taken at a magnification of 11,000 which show that the Paschen body is a brick-shaped structure with five areas of internal condensation.

THE IDENTITY OF ELEMENTARY BODIES AND THE VIRUS

Are the minute elementary bodies visualized by the various methods just described identical with the virus itself? There are many features which seem to prove that they are.

1. Ultrafiltration.—Early in the study of virus diseases it was learned that the causal organism could be separated from bacteria and tissue debris by forcing suspensions of the infectious material through diatomaceous earth or porcelain candles and asbestos filters of appropriate pore size. Viruses came to be known as "filtrable viruses." A great step in advance was made in 1929, when Elford [22] devised a method of preparing collodion membranes so that the pore size could be exactly determined in advance. Commercially these are known as Elford's "gradacol membranes." When a gradacol membrane of a pore size small enough to retain the virus is used to filter a suspension of vaccinia virus, no elementary bodies can be demonstrated in the filtrate. When inoculated into animals the filtrate is sterile. The retained material contains elementary bodies and is virulent on animal inoculation. Von Prowazek and de Beaurepaire,[23] using an agar filter, first demonstrated this for vaccinia in 1908, and it has been repeatedly confirmed.

2. Centrifugation.—Viruses are so small that centrifugation at high speeds is required to throw them down. Ledingham [24] demonstrated that if a suspension of vaccinia virus is centrifuged at 3,000 revolutions per minute for fifteen minutes

17. Haitinger, M.: Fluorescenz-Mikroscopie, Leipzig, Akad. Verlagsgesellschaft, m. b. H., 1938.
18. Hagemann, P. K. H.: Zentralbl. f. Bakt. (Abt. 1) **140**:184, 1937.
19. Barnard, J. E.: J. Roy. Micr. Soc. **56**:365, 1936.
20. von Borries, B.; Ruska, E., and Ruska, H.: Klin. Wchnschr. **17**:921, 1938.
21. Green, R. H.; Anderson, T. F., and Smadel, J. E.: J. Exper. Med. **75**:651, 1942.
22. Elford, W. J.: Brit. J. Exper. Path. **10**:126, 1929.
23. von Prowazek, S., and de Beaurepaire, H.: München. med. Wchnschr. **55**:2265, 1908.
24. Ledingham, J. C. G.: Lancet **2**:525, 1931.

tissue debris is thrown down. If the supernatant fluid is then centrifuged at 12,000 revolutions per minute in an angle head for one hour, the supernatant will contain no elementary bodies and will be sterile on animal inoculation, whereas the liquid at the bottom of the tube will contain large numbers of elementary bodies and will be virulent even in high dilutions. Other viruses have been similarly studied.

3. Size.—There are various methods for estimating the size of any given virus. Using careful microscopic measurements van Rooyen and Rhodes [25] found the diameter of the stained elementary body of vaccinia to be 0.200 micron. Elford and Andrewes [26] have estimated that the average pore size of a gradacol membrane which will allow the vaccinia virus to pass is 0.125 to 0.175 micron. The size of a virus can also be determined mathematically from the rate of deposit in a centrifuged specimen. The specific gravity of the virus must be assumed, however, in this method. Using this method, Elford and Andrewes [27] have estimated the size of the vaccinia virus to be 0.140 micron.

There exists, then, a close agreement on the size of the vaccinia virus as estimated by the various methods and that of the elementary body as measured microscopically. One should expect a stained elementary body to be larger than the average pore size which will permit its passage. The virus of vaccinia, for instance, is not a rigid sphere but is pliable and can squeeze through a smaller opening than its spherical diameter, just as a leukocyte can migrate through the narrow intercellular spaces of the epidermis. In addition, the amount of dye adsorbed may make the stained specimen appear slightly larger.

4. Specific Agglutination.—Von Prowazek and de Beaurepaire [23] in 1908 showed that the suspension of elementary bodies of vaccinia separated by ultrafiltration was specifically agglutinated by immune serum. Ledingham [24] prepared pure suspensions of vaccinia elementary bodies by high speed centrifugation and showed that the elementary bodies were specifically agglutinated by high dilutions of serums of animals rendered immune to vaccinia.

There would appear to be, then, strong evidence that the elementary body of vaccinia is identical with the virus. Similar evidence exists for other viruses. By analogy it may be presumed that in any disease in which elementary bodies can be visualized they are identical with the virus. It should not be assumed, however, that any given virus can be identified from the appearance of its elementary body. These are too small, and too near the limits of visibility, and the stains used are not differential or specific. However, neither can bacteria be identified by the microscopic appearance of stained specimens in most instances.

ELEMENTARY BODIES OF MOLLUSCUM CONTAGIOSUM

In 1905 Juliusberg [28] first demonstrated that the virus of molluscum contagiosum could pass through a Chamberlain filter and produce lesions on a volunteer. Wile and Kingery repeated this experiment and showed that the incubation period was fourteen to twenty-five days.[29] In 1907 Lipschütz,[30] who was a pioneer in the study of virus diseases of the skin, stained smears made from fresh material extruded from nodules and found innumerable minute spherical bodies which measured 0.250

25. van Rooyen, C. E., and Rhodes, A. J.: Virus Diseases of Man, London, Oxford University Press, 1940, p. 313.
26. Elford, W. J., and Andrewes, C. H.: Brit. J. Exper. Path. **13**:36, 1932.
27. Elford, W. J., and Andrewes, C. H.: Brit. J. Exper. Path. **17**:422, 1936.
28. Juliusberg, M.: Deutsche med. Wchnschr. **31**:1598, 1905.
29. Wile, U. J., and Kingery, L. B.: The Etiology of Common Warts, J. A. M. A. **73**:970 (Sept. 27) 1919.
30. Lipschütz, B.: Wien. klin. Wchnschr. **20**:253, 1907.

micron in diameter. He believed these were the causal agents and named them Strongyloplasma hominis. In 1911 he [31] fixed sections of molluscum nodules in sublimate alcohol and stained them with Giemsa stain. He found innumerable minute pink-staining bodies in the hypertrophied rete cells which he considered identical with those in the smear. He rightly believed them to be elementary bodies and the causal organism of the disease. These have subsequently been termed "Lipschütz bodies," just as the elementary bodies of fowlpox are called "Borrel bodies" and those of variola-vaccinia "Paschen bodies"—each term an eponym in honor of the discoverer. The elementary bodies of molluscum contagiosum share with those of psittacosis the distinction of being the largest known to infect the human being. Lipschütz [30] estimated their size to be 0.250 micron and van Rooyen [32] 0.3 to 0.35 micron.

To prepare a specimen, the contents of a molluscum lesion are expressed and collected on a slide. The material is crushed and spread thin with another slide. The elementary bodies are readily seen, unstained, with dark field illumination. If the fresh material is suspended on a slide in isotonic solution of sodium chloride and a cover slip applied, the unstained elementary bodies when viewed in the dark field can be seen crowding the molluscum bodies and showing vigorous brownian movement. In the dried and stained preparations enormous numbers of elementary bodies are seen especially along the track where the molluscum bodies have been drawn and around crushed molluscum bodies.

Findlay [33] filtered material from molluscum lesions through a Berkefeld V filter, centrifuged the filtrate at 15,000 revolutions per minute and obtained a sediment of nearly pure elementary bodies, which he inoculated successfully into the arm of a volunteer. This is the final proof that the elementary bodies are identical with the virus.

INCLUSION BODIES

An inclusion body in the sense the term is used by students of virus diseases is an abnormal product of the cell whose development has been stimulated by the presence of an active virus. Inclusion bodies may appear in the cytoplasm or in the nucleus or in rare instances in both. They may be acidophilic or basophilic. Not all viruses produce inclusions, nor are all bodies which appear to be inclusions evidence per se of virus infection.

THE INCLUSION BODIES OF VARIOLA-VACCINIA

The first inclusion bodies to be accurately studied were those described by Guarnieri [34] in the cytoplasm of cells invaded by the virus of variola or vaccinia. Weigert [35] had previously described them, but credit is given to Guarnieri for his intensive pioneer studies. They have been known since as Guarnieri bodies. He misunderstood their nature, however, and believed them to be active parasites, "cytorectes."

The easiest way to demonstrate Guarnieri bodies is by the inoculation of the cornea of a rabbit with vaccine virus. The eye is anesthetized with butacaine sulfate and cross hatched with a sharp needle, and a drop of active vaccinia virus suspension is rubbed in. In the next two days the bulbar conjunctiva becomes

31. Lipschütz, B.: Arch. f. Dermat. u. Syph. **107**:387, 1911.
32. van Rooyen, C. F.: J. Path. & Bact. **46**:425, 1928.
33. Findlay, G. M., in Doerr, R., and Hallauer, C.: Handbuch der Virusforschung, Berlin, Julius Springer, 1939, p. 302.
34. Guarnieri, G.: Arch. per le sc. med. **16**:403, 1892.
35. Weigert, C.: Anatomische Beiträge zur Lehre von den Pocken, Breslau, M. Cohn & Weigert, 1874.

injected, and faint, milky lines become visible with the loupe on the cornea. The rabbit is killed seventy-two hours after inoculation and the eye removed. The cornea is washed with sterile solution of sodium chloride and fixed in warm sublimate alcohol for two hours. Then it is hardened in alcohol and mounted in paraffin. Sections 4 to 5 microns thick are cut and stained with properly buffered

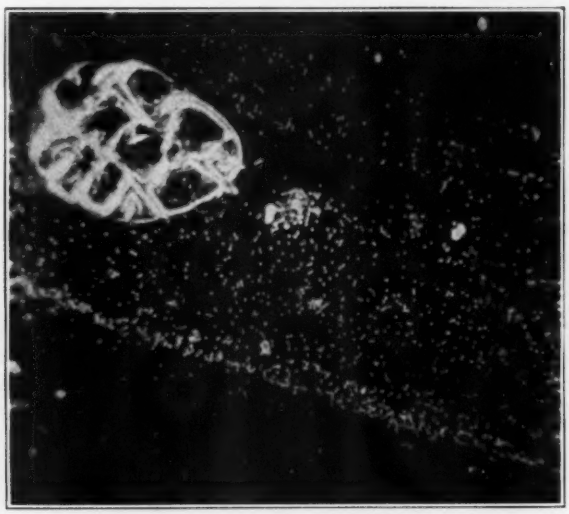


Fig. 6.—Elementary bodies (Lipschütz bodies) of molluscum contagiosum. Material expressed from a lesion was smeared on a slide, stained with Paschen's stain and photographed under oil immersion with dark field illumination. The remains of a crushed molluscum body are seen at one side of the field. The Lipschütz bodies are crowded along the line of direction of the smear.

Fig. 7.—Groups of staphylococci stained with Gram's stain and photographed with the same illumination and magnification as figure 6.

dilute Giemsa solution for twelve hours. In these sections, at the sites of inoculation there is a definite hypertrophy of the corneal epithelium, which is two to three times normal thickness. Descemet's membrane appears unchanged. There is little cellular reaction in the underlying connective tissue. The epithelial cells are two to three times larger than normal, and in the cytoplasm of many of them are seen one to three acidophilic staining bodies surrounded by a clear areola (fig. 8). These are the Guarnieri bodies, the cytoplasmic inclusions of vaccinia-variola. They are

amphophilic, with a slightly greater affinity for the acid dyes so that they appear pink with Giemsa stain, and are from 2 to 10 microns in diameter.

Torres [36] stated that the Brazilian form of alastrim can be distinguished histologically from variola vera by the fact that the cytoplasmic inclusions of alastrim in cutaneous lesions of man or experimental animals take a basophilic stain with Unna's hematoxylin and safranin while those of variola are acidophilic and stain bright red with this method.

ORIGIN AND NATURE OF GUARNIERI BODIES AND THEIR RELATION TO ELEMENTARY BODIES

In the past there has been much speculation and many theories to account for the appearance of Guarnieri bodies in the cytoplasm of cells inoculated with variola-vaccinia virus. They were variously considered to be protozoal parasites or to be composed of nuclear fragments, of extruded nucleoli or of chromatin particles

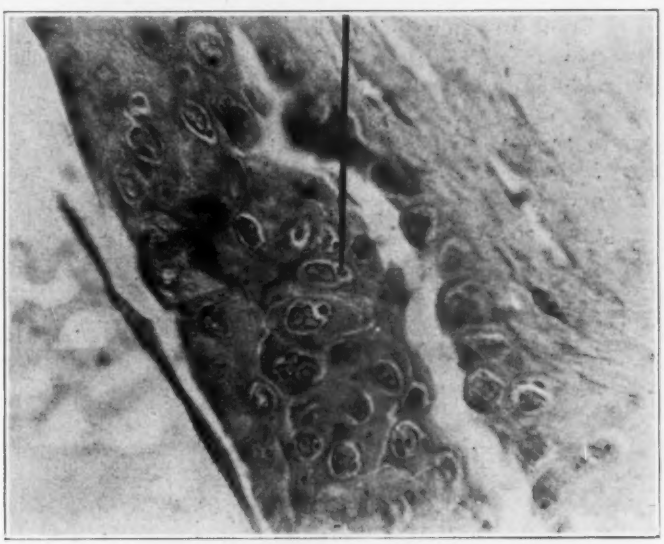


Fig. 8.—Guarnieri bodies. Section of a rabbit's cornea seventy-two hours after inoculation with vaccinia virus (Giemsa stain). The corneal epithelial cells are edematous. In a clear space surrounding a nucleus is seen the cytoplasmic inclusion body (indicated by the pointer). This stains pink; the nucleus stains blue. Black and white photography loses the contrast. The split between the basal layer and the outer epithelium is an artefact made by sectioning. Descemet's membrane shows no cellular infiltrate. Almost every epithelial cell in this area contains a Guarnieri body.

or to be engulfed leukocytes. Von Prowazek, however, staunchly maintained that they were "chlamydozoa," i. e., colonies of the elementary bodies surrounded by a mantle secreted by the cell. With the development of newer technics for the study of development of virus in tissue cultures and on the chorioallantoic membrane of the chick, the multiplication of the elementary bodies in the infected cell could be studied by direct or dark field illumination in living cells or in fixed and stained preparations. Still the whole subject remained confused. Haagen and Kodama [10] studied smears made from rabbits' corneas after inoculation with vaccinia and stained with victoria blue and found that several hours after infection some cells contained large numbers of elementary bodies and no Guarnieri bodies and others

36. Torres, C. M.: Proc. Roy. Soc. Med. **29**:1525, 1936.

contained Guarnieri bodies and no elementary bodies. They concluded that the multiplication of elementary bodies in the infected cell was independent of the Guarnieri bodies. The work of Goodpasture, Woodruff and Buddingh [37] contradicted this stand. They stained smears from chorioallantoic membrane of the chick embryo infected with vaccinia with acid fuchsin and found good evidence that the Guarnieri bodies contain masses of elementary bodies. Herzberg [38] used victoria blue stain on smears from infected chorioallantoic membrane. He expressed the opinion that the Guarnieri body was a form of virus colony, but not an obligatory phase, and that elementary bodies could multiply in the cell independently of the Guarnieri body. Himmelweit [7] devised an ingenious method for the examination of living cells of the chorioallantoic membrane of the duck by means of annular oblique incident illumination with a Leitz ultropak apparatus. He followed the multiplication of elementary bodies in living cells and concluded that the Guarnieri body seen in the stained preparations corresponds to localized irregular collections of elementary bodies and does not represent all the virus in the cell. Bland and Robinow [39] resolved the confusion by (1) using tissue cultures of rabbit's cornea which grew in thin sheets and could be handled readily, (2) using a purified suspension of vaccinia elementary bodies as an inoculum, (3) exposing the tissue cultures to the diluted inoculum for five minutes and washing off the excess. This made possible a determination of the exact time of inoculation which was impossible by other methods, (4) removing a certain number of the cultures every hour to be adequately fixed with osmic acid vapor and stained with Giemsa solution, (5) the statistical study of the time of appearance and number of each type of inclusion. They found that within an hour there appear small homogeneous bodies which are developed either by swelling of the elementary bodies or the deposit on them of some substances elaborated by themselves or by the cell. A little later, large homogeneous bodies appear, up to the size of a red blood cell. Slightly later, small networks appear which are more irregular in outline and stain less densely than the homogeneous bodies. At seven hours and continuing to twenty-four hours large networks appear. Elementary bodies are fairly numerous in the first few hours; they then disappear in the cells containing inclusions during the period of the small networks, only to reappear at the time of the appearance of the large networks. In exceptional cases they can be seen in the network. The authors conclude that the inclusion bodies are an obligatory stage in the multiplication of the virus and are colonies of elementary bodies enveloped in a matrix; this supports von Prowazek's original contention that the Guarnieri bodies are chlamydozoa. The inability to stain the elementary bodies in the network still requires explanation.

Nuclear inclusions have been described in the cutaneous lesions of variola and alastrim but never in vaccinia. They are eosinophilic and well developed in variola vera and less typical and more difficult to distinguish in alastrim, according to Torres.[36] He also stated that cytoplasmic inclusions and nuclear inclusions never occur together in the same cell.

THE INCLUSION BODIES OF MOLLUSCUM CONTAGIOSUM

The structure of a lesion of molluscum contagiosum has always been an interesting problem for the histopathologist. There occurs a benign acanthosis of the rete pegs with practically no reaction in the corium. The pear-shaped, hypertrophied rete pegs radiate from a central dell, which gives the appearance of

37. Goodpasture, E. W.; Woodruff, A. M., and Buddingh, G. J.: Am. J. Path. **8**:271, 1932.
38. Herzberg, K.: Zentralbl. f. Bakt. (Abt. 1) **136**:257, 1936.
39. Bland, J. O. W., and Robinow, C. F.: J. Path. & Bact. **48**:381, 1939.

invagination. The basal layer and two or three layers of cells above it appear unchanged. Some of the rete cells proceed to keratinize in the usual manner. The majority, however, undergo an interesting transformation as they are pushed outward. They lose their intercellular bridges and increase to eight or ten times normal size while the nucleus is pushed laterally and finally disappears. The cytoplasm becomes vacuolated and later homogeneous. The nature of the molluscum bodies thus produced has been much disputed. They have been variously considered as parasites, as a product of colloid, hyaline or keratinoid degeneration or as a peculiar variety of dyskeratosis. In 1911 Lipschütz,[31] after identifying the elementary bodies of molluscum contagiosum in fresh material, as already recorded, stained biopsy material from the lesions with dilute Giemsa stain. He described in molluscum bodies numerous minute spherical red-staining granules which he identified as identical with the elementary bodies demonstrated in fresh smears. He advanced the opinion that the molluscum body is an inclusion body and analogous to the Guarnieri body of variola-vaccinia. Since the elementary bodies are surrounded by a mantle of cellular material, he called them chlamydozoa.

In 1927 Goodpasture and King[40] reinvestigated the cytology of molluscum contagiosum, for they considered the nature of the molluscum body a basic problem in the cellular pathology of virus infection. They were particularly interested in the role played by the extruded nucleoli and mitochondria in the development of the molluscum body. They found that the basal layer is relatively normal except that the cells are larger and in more active mitotic division. As the rete cells are pushed peripherally, the majority undergo a series of changes. The extrusion of nucleoli and increase in mitochondria which occur in the lower rows of cells take no part in the formation of the inclusion body. The cells enlarge as they are pushed outward; the central portion becomes basophilic, and vacuoles appear peripherally. A few acidophilic granules appear about the borders of the vacuoles and eventually fill them. The basophilic cytoplasm becomes condensed as a network between these granular vacuoles. The nucleus is pushed aside and flattened. The eosinophilic granules are elementary bodies of Lipschütz. As the cells approach the surface, the central mass shrinks away from the thin membrane of cytoplasm and fuses to form the molluscum body lying within a framework consisting of its own cell membrane and the remnant of the few surrounding cells which have keratinized normally. The molluscum bodies are thus formed by desiccation and not by keratinization. Goodpasture and King considered the changes consistent with the growth and multiplication of an active virus in the cell.

Finally in 1934 van Rooyen,[32] using a Chambers microdissection apparatus, was able to show that the molluscum body, which is egg shaped, has a little cap at the small end. When this is punctured, large numbers of elementary bodies are extruded. He identified the mantle chemically as carbohydrate. We have frequently demonstrated brownian movement of the Lipschütz bodies in fresh molluscum bodies suspended in isotonic solution of sodium chloride. In stained smears made from fresh material, myriads of elementary bodies appear along the line where the molluscum body has been crushed (fig. 9).

Thus it has been conclusively demonstrated that the extraordinary molluscum body, which caused so much speculation, is really an overgrown cytoplasmic inclusion due to the action of the virus; that it has shrunk away from remnants of the cytoplasm, and that it houses myriads of elementary bodies, doubtless the virus, which are being constantly extruded onto the surface of the skin, where they are the source of infection for the patient himself as well as for others. The Guarnieri

40. Goodpasture, E. W., and King, H.: Am. J. Path. **3**:358, 1927.

body of variola-vaccinia houses the Paschen bodies, just as the molluscum body houses the Lipschütz bodies. However, it would be unwise to conclude from these examples that all virus inclusions are formed in a similar fashion. Nonetheless, Malamos [41] has shown good evidence that the cytoplasmic inclusions of lymphogranuloma venereum are aggregates of elementary bodies in a mantle produced by the cell.

COMPARISON OF VARIOLA-VACCINIA WITH MOLLUSCUM CONTAGIOSUM

Variola-vaccinia has some similarities to molluscum contagiosum and many striking differences. The elementary bodies of both are large and readily demonstrated. The inclusion bodies of both are cytoplasmic and eosinophilic and are an obligatory phase in the multiplication of the virus in the invaded cell. The Guarnieri body and molluscum body have both been shown to contain a great number of elementary bodies when mature. These elementary bodies are the source of new infection.

The virus of molluscum contagiosum is strictly epidermotropic. While the obvious clinical manifestations of variola-vaccinia are on the skin, here the

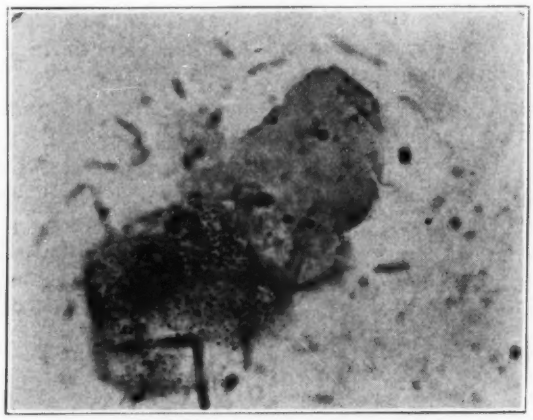


Fig. 9.—Lipschütz bodies in crushed molluscum body. Material and stain the same as in figure 1. Photographed under oil immersion and ordinary illumination.

resemblance ceases. Using the chorioallantoic membrane of the chick, Goodpasture, Woodruff and Buddingh [37] have shown that the vaccinia virus involves ectoderm, mesoderm and entoderm. They demonstrated Guarnieri bodies in the epidermis, connective tissue cells and histiocytes, in the endothelium and advential cells of the blood vessels and in the entodermal epithelium.

Molluscum contagiosum is anchored in the epidermis. Variola-vaccinia is a systemic infection. Numerous investigators have found the blood and tissue fluids of variola infectious. Even prophylactic vaccination as ordinarily practiced causes virus to appear in the blood of a nonimmune subject. By means of animal inoculation, Gins, Hackenthal and Kamentzewa [42] were able to demonstrate the presence of vaccinia virus in the tonsils of children three days after successful vaccination. The same authors were able to demonstrate the presence of vaccinia virus in all the internal organs of rabbits and guinea pigs inoculated with this virus intradermally five days previously. As already stated, Haagen and Kodama [10] demonstrated by

41. Malamos, B.: Zentralbl. f. Bakt. (Abt. 1) **143**:1, 1938.

42. Gins, H. A.; Hackenthal, H., and Kamentzewa, N.: Zentralbl. f. Bakt. (Abt. 1) (no. 6-8, supp.) **110**:115, 1929.

means of a victoria blue stain the elementary bodies of vaccinia in smears made from the internal organs of experimentally infected animals. There is no question that prophylactic vaccination produces a systemic infection just as truly as smallpox does.

Since variola and vaccinia are systemic infections, immune processes are so stimulated that the disease is self limited and a more or less permanent immunity follows. Virus-neutralizing antibodies, complement-fixing antibodies, agglutinins and precipitins are readily demonstrated in the blood serum after an attack of variola or after a successful vaccination. The virus of molluscum contagiosum never enters the circulatory system. There are no systemic manifestations. Production of immune bodies is not stimulated. No one has ever demonstrated immune bodies in the serum of patients with this disease. Consequently the disease is not self limited. Instead, it is autoinoculable and may persist indefinitely. There is, without question, considerable natural cellular immunity to the virus in the human epidermis. Only a few persons become infected, and mere puncture of the lesion seems to stimulate enough resistance to cure it.

The usual portal of entry for variola virus is the respiratory tract.[43] Guarnieri bodies have been demonstrated in the epithelium of the lungs and upper respiratory tract. The elementary bodies are excreted with the breath. They are so minute that they remain suspended in the air for long periods. Elementary bodies are also present in large numbers in crusts, scales and secretions of the skin and on objects handled by the patient. A nonimmune person is usually infected by inhalation of these elementary bodies, which can enter the circulation by way of the nasal mucosa as well as by way of the lungs. Variola virus may also be inoculated into the skin, as it was for centuries by the Chinese, the earliest protective vaccination with human material. The port of entry of vaccinia virus is usually through a superficial break in the stratum corneum. The virus may be introduced intentionally or accidentally. It may also be introduced by subcutaneous or intravenous injection or by a nasal spray. Whatever the portal of entry, if the inoculation is successful virus appears in the blood and a systemic infection results. In molluscum contagiosum the elementary bodies are being constantly excreted into the skin of the patient through the central dell. The portal of entry for a new infection is undoubtedly a slight break in the stratum corneum. The spread of the disease in Turkish baths and massage parlors would indicate that mediate as well as immediate contact with a source of infection may spread the virus.

Molluscum contagiosum is an obligate parasite on human epidermis. Unlike variola-vaccinia, it has never been transferred to lower animals, to tissue cultures or to the chorioallantoic membrane of the chick. Possibly it could be cultivated on tissue cultures of human epidermis, but so far there is no record that this has ever been done.

SUMMARY

The photomicrographs presented demonstrate the elementary bodies of variola, vaccinia and molluscum contagiosum as well as the relationship of the elementary bodies of molluscum contagiosum to the molluscum body. The experimental production of the cytoplasmic inclusion bodies of variola-vaccinia, known as Guarnieri bodies, is also illustrated by a photomicrograph; modern research indicates that they are an obligatory phase in the multiplication of the virus in the cells.

There is evidence that the "molluscum body" is really an exaggerated inclusion body housing vast numbers of the elementary bodies.

Interesting similarities and differences exist between variola-vaccinia and molluscum contagiosum.

43. Friedemann, U., and Gins, H. A.: Deutsche med. Wchnschr. **43**:1159, 1917.

ACANTHOSIS NIGRICANS

IS IT A FORM OF AVITAMINOSIS?

LESTER HOLLANDER, M.D.
PITTSBURGH

Acanthosis nigricans is a puzzling and rare but distinctive disease of the skin and the mucous membranes. Of its characteristics, it is the striking appearance which has occasioned the many observations which have been made and reported. The characteristic features are changes in pigmentation, both increases and decreases; the appearance of vegetative warty growths; dystrophy and even loss of hair and nails; papillation [1] about the mucocutaneous junctions of the mouth, eyelids, external genitalia and perianal area, and itching of variable character, which at times is unbelievably intense.

As a general rule, two types are recognized. One is the benign juvenile form, which occurs in children and which is usually of little consequence, as it bears no relation to the general well-being. The other is the adult type, which in the vast majority of instances occurs in cachetic persons who suffer from carcinoma of the stomach or intestines, sarcoma, severe diabetes or tuberculosis—malignant diseases always ending fatally in a relatively short time.

There are many puzzling questions which confront one, for instance: whether the juvenile and the adult type are in any way associated with each other, since they occur under such entirely different circumstances; why the adult type occurs so rarely when the cachexia of severe malignant diseases is relatively frequent, and why such divergent diseases as carcinoma, sarcoma and diabetes produce a rare but distinctive disease such as acanthosis nigricans.

It was Pollitzer [2] who, in 1884, first described acanthosis nigricans; he was followed by Janovsky.[3] Unna [4] in 1889 named the disease acanthosis nigricans. Other names employed were *dystrophie papillaire et pigmentaire,* by Darier,[5] keratosis nigricans, by Crocker,[6] and *dystrophie papulo-pigmentaire,* by Hallopeau.[6]

Interesting observations on this disease have been made. Masson and Montgomery [7] stated that acanthosis nigricans is probably attributable to a lesion or functional disturbance of the abdominal sympathetic system and that it is more common than published statistics indicate. Petrini de Galatz [8] described a patient suffering from acanthosis nigricans associated with a pulmonary carcinoma and refuted the commonly accepted hypothesis that the cutaneous changes occur secon-

From the Pittsburgh Skin and Cancer Foundation.

1. Sutton, R. L., and Sutton, R. L., Jr.: Diseases of the Skin, ed. 10, St. Louis, C. V. Mosby Company, 1939, p. 1507.

2. Pollitzer, in Internationaler Atlas seltener Hautkrankheiten, Leipzig, 1890; Acanthosis Nigricans: A Symptom of Disorder of the Abdominal Sympathetic, J. A. M. A. **53**:1369 (Oct. 23) 1909.

3. Janovsky, in Internationaler Atlas seltener Hautkrankheiten, Leipzig, 1890.

4. Unna, cited by MacLeod, J. M. H.: Diseases of the Skin, New York, Paul B. Hoeber, 1921, p. 1100.

5. Darier, J.: Ann. de dermat. et syph. **4**:865, 1893.

6. Cited in Yearbook of Dermatology and Syphilology, Practical Medicine Series, Chicago, Year Book Publishers, Inc., 1914, vol. 9, p. 13.

7. Masson, J. C., and Montgomery, H.: Am. J. Obst. & Gynec. **32**:717, 1936.

8. Petrini de Galatz: Ann. de dermat. et syph. **5**:321, 1914.

dary to a compression of the abdominal sympathetic nerves by an abdominal neoplasm, as his patient's carcinoma did not involve such an area. Wise [9] reported a case of acanthosis nigricans following decapsulation of the kidneys in a young woman, 25 years of age, and thought that he was justified in assuming that this dermatosis is a manifestation directly related to disturbances or impairments of function in the abdominal cavity in some manner provoked by the decapsulation of the kidneys. Knowles, Sidlick and Ludy [10] expressed the belief that excitation of toxemia, generated by such diseases as carcinoma, tuberculosis and syphilis, and sunlight, acting on the enzymes in the skin, must be considered as causal factors.

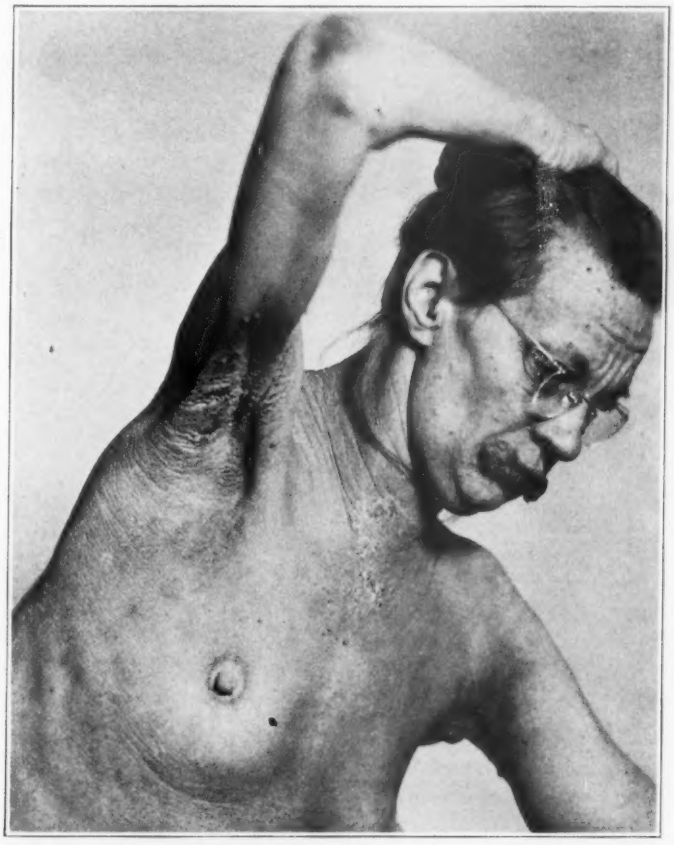


Fig. 1.—Cordlike thickenings in the axillas; variety of warty excrescences and discoloration on the neck; depigmentation about the nipple and areola, and papillary excrescences about the mouth.

There are investigators who tend to believe that acanthosis nigricans results from endocrine dysfunction. Helen Curth [11] stated that changes in the endocrine system play a role and who further postulated that there appears to be a genetic relationship between acanthosis nigricans and cancer but that precisely what this

9. Wise, F.: J. Cutan. Dis. **36**:35, 1918.

10. Knowles, F. C.; Sidlick, D. M., and Ludy, J. B.: Acanthosis Nigricans: Adult and Juvenile Types, Arch. Dermat. & Syph. **19**:391 (March) 1929.

11. Curth, H.: Benign Type of Acanthosis Nigricans: Etiology, Arch. Dermat. & Syph. **34**:353 (Sept.) 1936.

relationship is is still unknown. Hellerström [12] reported a case of familial juvenile acanthosis nigricans associated with severe diabetes mellitus in which both the cutaneous disease and the diabetes improved greatly after total castration. He expressed the belief that the endocrine character of acanthosis nigricans was demonstrated by its retrogression after castration. Grace and Schwartz,[13] on the other hand, could discover no definite evidence of endocrine disturbance in their studies and held the hypothesis of endocrine dysfunction as an etiologic agent in acanthosis nigricans to be totally unfounded.

Fig. 2.—Papillary excrescences about the palpebral margins and covering the surfaces of the lips and deep fissures on the tongue.

Darier [14] and Bloch [15] assumed that acanthosis nigricans results from some disturbance of the adrenal glands, which have often been found invaded by tumor

12. Hellerström, S.: Acta dermat.-venereol. **14**:86, 1933.

13. Grace, A. W., and Schwartz, H. J.: Acanthosis Nigricans: Case of Benign Form in Adult Investigated from Aspect of Endocrine Dysfunction, Arch. Dermat. & Syph. **29**:691 (May) 1934.

14. Darier, J.: Ann. de dermat. et syph. **4**:421, 1893.

15. Bloch, cited by Ewing.[16]

cells; but Ewing [16] stated that such a theory is not likely, because in many cases the pigmentary changes begin before the appearance of the visceral cancer.

These references are discussed merely to emphasize the variety of opinions which exist in regard to the causation of acanthosis nigricans. To them I wish to offer for consideration another probability reasonably assumed as a factor in the adult type. I consider this disease to be the result of a severe avitaminosis which occurs because these persons are unable to consume and absorb required quantities of vitamins, at times as the result of lack of gastric capacity and at times as the result of lack of proper composition of digestive fluids. Thus, the occurrence of acanthosis nigricans with such a variety of malignant disturbances as carcinoma, sarcoma, diabetes and tuberculosis becomes somewhat more explainable. The appearance of the skin, the thickening, the scaling, the changes in pigmentation and the presence of papillary elevations have been accounted for by vitamin

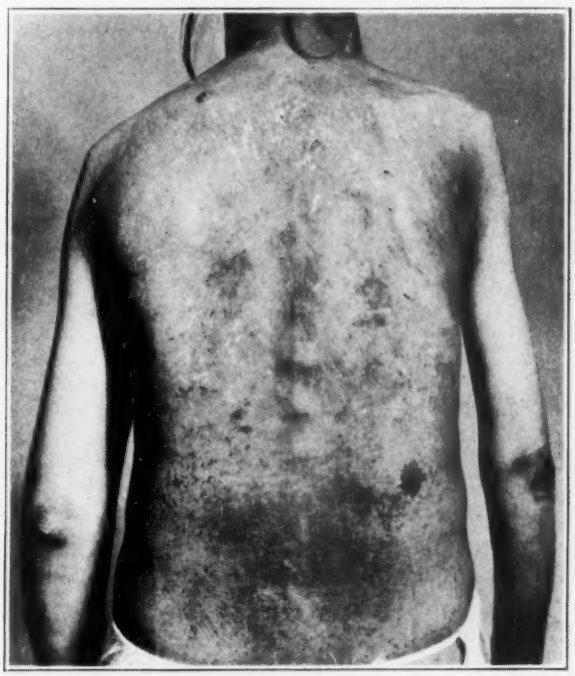


Fig. 3.—Dirty grayish pigmentation of back and depigmentation of scars.

deficiency in connection with other cutaneous disturbances. One of the observations in the laboratory examination of the patient to be described was the complete absence of demonstrable vitamin C in the blood stream. It is true that furnishing large doses of vitamin C did not improve the appearance of her skin, but massive injections of vitamin B complex made an appreciable change in the papillary excrescences about the mouth and the mucocutaneous junction of the eyelids.

I believe that the case reported by Alkiewicz [17] helps to substantiate this contention of avitaminosis. His patient, who had acanthosis nigricans associated with multiple necrotic periarteritis nodosa, suffered from extensive hemorrhages of the skin and mucous membranes. Such a symptom naturally leads one to think of the lack of antiscorbutic factors in the diet.

16. Ewing, J.: Neoplastic Diseases, ed. 4, Philadelphia, W. B. Saunders Company, 1941. p. 891.

17. Alkiewicz, J.: Arch. f. Dermat. u. Syph. **168**:522, 1933.

REPORT OF A CASE

Mrs. S. P., a 38 year old housewife of Slavish extraction, was referred to the Pittsburgh Skin and Cancer Foundation on May 11, 1943, on account of an extensive and unusual eruption which distorted the entire surface of the skin.

It began about February 1942, with a generalized itching and the development of some indefinite epigastric pain. Of these the itching was the more intolerable, and, although she consulted several physicians, nothing of any importance was discovered nor was relief obtained. In about June 1942, she noticed that the skin of her hands became rough and also that pigmentary changes—deep coloring of her skin—appeared over several surfaces, especially the axillas, abdomen, neck and back. Later, excrescences of a warty nature appeared, and as the visible surface changes increased the itching decreased. However, she began to lose weight, became tired and weak and lost her appetite, and the pain in her abdomen became more acute and of longer duration.

Her previous medical history furnished no important detail. She was married and had three living children; she had had one miscarriage. Her weight was 115 pounds (52.1 Kg.).

General and roentgenologic examinations revealed the presence of a gastric carcinoma in the form of an infiltration along the great curvature of the stomach extending from the middle of the corpus down to the proximal edge of the pyloric valve. The infiltration had almost completely encircled this portion of the stomach.

Fig. 4.—Complete loss of hair of the mons veneris; triangular pigmentary changes, and papillomatous excrescences arising from the labia minora.

The entire cutaneous surface was rough, dry and dirty looking on account of hyperpigmentation and depigmentation and the presence of the warty excrescences. At specific locations the changes in the skin were sufficiently different to merit detailed descriptions.

The backs of the hands were covered by brownish black and grayish black flat-surfaced warty elevations. The nails were shiny and dry. The palms had lost their soft and even surface and had raised furrows, giving the appearance of cloth made of whipcord. Otherwise, the skin was dry and had lost its pliable character.

On the flexor surfaces of the arms and forearms there were areas of brownish black pigmentation, becoming distinctive and more sharply circumscribed and forming oval areas about the elbows. The rugae of the normal skin were intensified.

Both axillas were covered by darkly pigmented skin, which was composed of thickened and raised rugae forming furrows of various depth, the base of which seemed depigmented by contrast.

The skin of the neck was pigmented, various shades of black, brown and tan being discernible, and there were warty lesions present in the form of black, grayish brown and grayish white digitate warts. The latter gave the appearance of being edematous and inflamed. They stood out prominently.

The face gave the appearance of being dirty, as the pigment was unevenly distributed. The conjunctivas were red and inflamed. Grayish white warty excrescences covered the mucocutaneous junction of the palpebral slits. Beginning at the vermilion border and covering the entire mucous membrane–covered portion of the lips, warty vegetations were present. These were thickly studded and were most pronounced at the angles of the mouth. On the

upper and lower alveolar mucous membranes at the incisor area pinkish red papillation was present. The tongue itself was deeply fissured at irregular intervals. Furthermore, it was shiny and dry.

On the anterior portion of the torso the median line was pigmented, and this pigmentation extended from the ensiform cartilage to the mons veneris. An areola of pigment about the umbilicus accentuated the warty excrescences in this location. Both the nipples and the areolas surrounding them were depigmented, dry and inelastic.

On the back the skin was discolored, with an abortive attempt at symmetry. Scars of previously experienced traumas, as they were depigmented, appeared glistening white.

The mons veneris was completely devoid of pubic hair, and the area was sharply defined by irregular hyperpigmentation. The labia minora were thickened as the result of a heavy warty growth formation similar to that observed about the lips and palpebral slits.

About the perianal area the warty excrescent growth was the most profuse, and it formed a double-ringed rosette-like protrusion of a uniform gray color.

The popliteal spaces showed a condition similar to that of the axillas, while the cutaneous picture of the feet resembled that described of the hands.

Microscopic examination was made of sections obtained from the axilla and from the papillary excrescences about the mouth, confirming the usual architecture of acanthosis nigricans: increased pigment content of the malpighian layer, acanthosis and hyperkeratosis.

Otherwise, there was nothing distinctive in any further examination.

The course of this disease was followed for approximately four weeks. The patient was given daily injections of vitamin B complex in 2 cc. doses, and there occurred an appreciable decrease of the vegetative growths about the eyelids and lips. However, she became extremely weak and could not attend the outpatient department. She was hospitalized in an adjoining suburban town, where she died on July 21, 1943. An autopsy was performed, and an extensive adenocarcinoma of the stomach was found.

SUMMARY

The patient whose case is reported had extensive involvement of her skin with acanthosis nigricans. This may have been a manifestation of a vitamin deficiency.

631 Jenkins Building.

Clinical Notes

ALLERGIC DERMATITIS CAUSED BY HAIR LACQUER PRODUCTS

ALFRED HOLLANDER, M.D., SPRINGFIELD, MASS.

The following communication is concerned with dermatitis originating from the use of hair lacquer pads and hair lacquer, based on my observations in 15 cases, in 11 of which the eruption was caused by hair lacquer pads and in 4 by hair lacquer itself.

The firm manufacturing the hair lacquer pads informed me that their hair lacquer is composed of two types of synthetic resin. The firm manufacturing the hair lacquer reported that their product consists of a mixture of shellac and myrrh.

The first symptoms of dermatitis appeared after the offending agent had been used for one to three days. The main complaint was severe pruritus.

The fully developed picture showed an erythematopapular eruption along the entire hair line, with considerable edema of both eyelids, ears and temporal regions. Twenty-four to forty-eight hours after the onset of the symptoms the erythematopapular eruption and edema expanded over the entire face, neck and upper part of the chest. A few discrete papules were scattered over both arms, preferring the cubital fossae, and over the anterior aspect of both thighs. Some vesiculation was observed in 2 cases.

All lesions subsided after three to seven days. The edema of the eyelids and ears was persistent and disappeared only after ten to fourteen days.

Patch tests with both hair lacquer pads and hair lacquer were performed on all patients. All of them showed a positive reaction after twenty-four to forty-eight hours.

Patch tests with 1 per cent sodium hydroxide and 0.06 per cent ammonia performed on 6 patients yielded negative reactions.

An interesting additional observation was the fact that 12 patients showed a positive reaction to the adhesive tape used to keep the test patches in place. Because the adhesive tape also contains a resin, I anticipated the same positive reaction to both lacquer and adhesive tape in the same patients and therefore placed a piece of gauze over and beyond the tested area so that a ½ inch (1.25 cm.) wide free space was obtained between the lacquer and the adhesive tape. The free space remained normal.

From this observation it can readily be assumed that resins contained in both hair lacquer pads and hair lacquer are the etiologic factor which caused this type of allergic dermatitis. The positive reaction to adhesive tape confirms the conclusion that there exists a definite allergy to this group of resins.

Since resins, both natural and synthetic, are powerful sensitizers, manufacturing firms using these and similar materials should be compelled, if necessary by legislation, to have toxic and dermatologic tests performed with these chemicals. This same suggestion has been made previously by Dr. Louis Schwartz and his associates in regard to an outbreak of dermatitis caused by new resin fabric finishes.

Dr. Louis Schwartz, Chief, Dermatoses Investigations Section, United States Public Health Service, to whom I reported my observation, informed me that he had received complaints of dermatitis caused by hair lacquer pads. The reports of similar cases came to him from Dr. Isaac R. Pels, Baltimore; Dr. Ralph Bowen, Houston, Texas, and Dr. William D. Whitehead, Scranton, Pa.

458 Bridge Street.

VON RECKLINGHAUSEN'S DISEASE WITH INVOLVEMENT OF THE SCALP, PALMS AND SOLES

Report of a Case

GERALD A. SPENCER, M.D., NEW YORK

Although von Recklinghausen's disease is systemic, involving not only all parts of the skin but the visceral organs, I am presenting the following case because of the unusual involvement of the scalp, palms and soles.

REPORT OF CASE

A. W., a Negro woman aged 31, was referred to me for the removal of a painful growth in the region of the right collar bone.

She stated that at about the age of puberty she noticed the appearance of several small growths on her arms. From then on, they became more numerous and extended to all parts

A, small tumors on the face and neck with large tumors on the scalp; *B*, few discrete tumors in the palm; *C*, multiple discrete pinhead-sized to pea-sized tumors and a large pedunculated tumor.

of her body; within recent years, she had noticed their appearance on her scalp and limbs. She had been subject to dizziness from the age of 21 years and had been having frequent attacks of headache, which occurred as often as two or three times a month. There had been no serious acute illness, and she had never been pregnant. Both mother and father were dead, but neither of them had had a similar disease of the skin. They had had eight

children, only three of whom were still alive; one living brother had similar growths on his body.

The physical examination revealed a fairly intelligent woman whose body was covered with discrete soft tumors ranging from the size of a pea to that of an egg. Some of them, besides being soft, were pedunculated. In the region of the right side of the clavicle there was a hard egg-sized tumor, which was painful on examination and for which she sought surgical removal. Few hyperpigmented spots were visible on the trunk. On the face and especially in the scalp were several discrete soft tumors ranging from the size of a pea to that of a grape (fig. *A*). The palms also showed a few hard pea-sized lesions (fig. *B*). On the soles, especially in the region of the arches, there were similar discrete lesions, one of which was unusually large and pedunculated (fig. *C*). The rest of the physical examination gave normal results.

Laboratory examination showed the urine to be normal for sugar and albumin. The Wassermann reaction of the blood was negative. The blood count showed 4,190,000 erythrocytes, a hemoglobin content of 86 per cent, and 4,500 leukocytes, with the following differential count: neutrophile polymorphonuclear cells 50 per cent, eosinophile cells 4 per cent and lymphocytes 45 per cent.

Roentgen examination of the skull showed areas of hyperostosis in the frontal and parietal bones.

2135 Seventh Avenue.

Correspondence

THE CHICK EMBRYO ANTIGEN (LYGRANUM) TEST FOR LYMPHOGRANULOMA VENEREUM

To the Editor:—Dr. Heisel and his collaborators[1] state that "the originators of the test [used] a 7 mm. papule as a minimal positive [Frei] reaction. . . ." In the report which introduced chick embryo antigen (lygranum) as a material for the Frei test[2] it was noted that "the upper limit of the diameter of non-specific papules produced by these materials [lygranum antigens and control] was 5 mm." In the table accompanying that report it is clear that a papule 6 mm. in diameter was regarded as a positive reaction. Support has been given to the selection of this size of papular reaction when lygranum is used as the minimum which can be called positive by the observation[3] that in 24 persons in whom the diameter of the papule of the Frei reaction was 6 mm. and who either presented clinical manifestations which could be regarded as lymphogranulomatous or had evidence of infection with another venereal disease the complement fixation reaction for lymphogranuloma was positive in dilutions of serum ranging from 1:6 to 1:120. No instance has yet been encountered in which a papular response of 6 mm. diameter to the cutaneous test when lygranum was employed was accompanied by a negative complement fixation reaction.

No tears need be shed over the passing of human pus antigens; to repeat the convincing arguments against the continuation of their use[4] would be to flog a long dead horse.

Dr. Heisel's statement that "it is unfortunate that the difference between a positive and a negative result must be measured in terms of tenths of a centimeter" is simply an expression of the limitations of Frei's cutaneous test, in which it is difficult at times to be sure of the exact dimensions, in millimeters, of the papular response. This lack of certainty becomes important when the size of the papule approaches the borderline between positive and negative and has undoubtedly been responsible for many errors in diagnosis. The frequency of occurrence of such errors can now be greatly reduced by the use of the complement fixation test (which ought more conveniently, and with due credit, to be called the Rake test),[5] which possesses limits of uncertainty narrower than those of the Frei test and is more sensitive. It has been my custom during the past three years to have a complement fixation test performed on all persons who present clinical manifestations which could be regarded as lymphogranulomatous and in whom the Frei reaction is either doubtful or negative. My experience with the complement fixation test has been most satisfactory and has strengthened the opinion expressed soon after its introduction[6] that "it will come to be regarded as the final court of appeal in determining the lymphogranulomatous status of a clinical condition."

ARTHUR W. GRACE, M.D., Brooklyn.

11 Schermerhorn Street.

1. Heisel, E. B., and Stroud, G., III, in collaboration with Krause, M.; Hubler, W.; Cole, H. N., Jr., and Cord, R.: The Chick Embryo Antigen (Lygranum) Test for Lymphogranuloma Venereum: A Clinical Investigation, Arch. Dermat. & Syph. **48**:379 (Oct.) 1943.

2. Grace, A. W.; Rake, G., and Shaffer, M. F.: A New Material (Lygranum) for Performance of the Frei Test for Lymphogranuloma Venereum, Proc. Soc. Exper. Biol. & Med. **45**:259, 1940.

3. Grace, A. W., and Rake, G.: Complement Fixation Test for Lymphogranuloma Venereum: Results Obtained with Its Use, Arch. Dermat. & Syph., this issue, p. 619.

4. Grace, A. W., and Suskind, F. H.: The Use of Standardized Mouse Brain Antigen for the Performance of the Frei Test for Lymphogranuloma Inguinale, J. A. M. A. **107**:1359 (Oct. 25) 1936.

5. McKee, C. M.; Rake, G., and Shaffer, M. F.: Complement-Fixation Test in Lymphogranuloma Venereum, Proc. Soc. Exper. Biol. & Med. **44**:410, 1940.

6 Grace, A. W.: Lymphogranuloma Venereum, Bull. New York Acad. Med. **17**:627 (Aug.) 1941.

Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

PHOTODYNAMIC EFFECTS IN DERMATOLOGY. JOHN H. STOKES, HERMAN BEERMAN and NORMAN R. INGRAHAM JR., Am. J. M. Sc. **204**:601 (Oct.) 1942.

The authors review the diseases with a prominent cutaneous aspect in which photodynamic effects appear to play a leading part. There are numerous references to the literature on hydroa vacciniforme, the porphyrias, chronic polymorphic eruptions due to light, pellagra, urticaria solaris, berlock dermatitis and photodermatitis due to meadow grass, the relation between sunlight and cancer, xeroderma pigmentosum, lupus erythematosus and other dermatoses with light sensitization factors. There is no editorial comment, but the work represents a most extensive compilation of references. The discussion of treatment is brief, though numerous recommended measures are listed. The authors offer no opinion as to their value but provide a number of formulas for protective applications.

LYNCH, St. Paul.

TREATMENT OF OTOMYCOSIS. MERRILL J. REEH, Ann. Otol., Rhin. & Laryng. **51**:146 (March) 1942.

All debris must be removed before treatment is started. The medicament is best applied on a wick for the first two or three days, the wick being left in place for twelve to twenty-four hours at a time. Various medicaments were used in this study, but the best results were secured from the use of metacresyl acetate and of metacresyl acetate containing 1 per cent thymol, the mixture being superior to the metacresyl acetate alone. After the wick had been in place twelve to twenty-four hours, the walls of the canal were white, dry and exfoliative, and much of the dry skin and debris could be readily removed with an applicator. If the solution proved too strong, it was diluted with olive oil. Occasionally drops of the medicament, used after the wicks had been discontinued, were diluted.

MILLER, Philadelphia. [ARCH. OTOLARYNG.]

[I] CONGO RED TEST FOR AMYLOID DISEASE: A QUANTITATIVE TECHNIC. [II] UTILITY OF THE CONGO RED TEST IN DIAGNOSIS AND IN DIFFERENTIAL DIAGNOSIS. PAUL H. HARMON and GRAHAM KERNWEIN, Arch. Int. Med. **70**:416 and 421 (Sept.) 1942.

Harmon and Kernwein report the results of 92 tests with congo red made by a quantitative technic which depends on the administration of a dose graded according to the patient's weight. The samples of blood were removed at four minutes and at one hour after injection in all cases and also at nine and fourteen minutes in cases in which a palpable liver or spleen suggested that the amount of amyloid was large. Repetition of this test at various stages of amyloid disease allowed demonstration of accumulation of stores of amyloid in the body in some cases and disappearance of deposits of amyloid in cases in which recovery was noted.

In their second article Harmon and Kernwein report that the minimum time for the accumulation of sufficient amyloid to give a positive reaction to a dye test is nine months and the average is one and one-half to two years. They also point out that since slight to moderate amounts of amyloid may be present and the test will fail to reveal this fact, a negative reaction is not positive proof that amyloid is not present.

HODGKIN'S DISEASE WITH SPECIFIC LESIONS APPEARING FIRST IN THE SKIN. HOBART A. REIMANN, W. PAUL HAVENS and PETER A. HERBUT, Arch. Int. Med. **70**:434 (Sept.) 1942.

Reimann, Havens and Herbut describe a case in which the first evidence of Hodgkin's disease appeared as specific lesions in the skin and fever. Two months elapsed before there was clinical evidence of visceral involvement. The lesions appeared as nodules, most of which disappeared and left brownish spots; others ulcerated and became crusted. The histologic changes were characteristic but were not pathognomonic enough to confirm the suspected diagnosis.

LYNCH, St. Paul.

LEAD POISONING CAUSED BY PLASTER OF LEAD OLEATE (DIACHYLON). MYRON H. MATZ, Arch. Pediat. **59**:805 (Dec.) 1942.

The author reports a case of lead poisoning occurring in a white girl 20 months of age, who in the course of treatment for infantile eczema acquired lead poisoning from the application of plaster of lead oleate. The symptoms were attributed to poisoning from the use of plaster of lead oleate because of (1) the basophilic stippling of red blood cells, (2) the changes consistent with lead poisoning shown in roentgenograms of the long bones and (3) the absence of any other probable causes.

In reviewing the literature the author found that lead poisoning resulting from the use of plaster of lead oleate is rare. Only 3 cases have been reported, and the case reported by H. Passler in 1894 was almost identical with this case. GELBER, Los Angeles.

"FIELD-FIRE" AND INVASIVE BASAL CELL CARCINOMA—BASAL-SQUAMOUS TYPE. JAMES B. BROWN and FRANK MCDOWELL, Surg., Gynec. & Obst. **74**:1128 (June) 1942.

The authors have observed a type of basal cell carcinoma that spreads through the derma without causing much surface ulceration and without producing much new tissue. It seems to be almost entirely destructive and to leave behind a healed area, but to present an ever increasing lesion with a small raised, advancing edge possessing intact or temporarily ulcerated epidermis. Microscopic sections from the edge and from any remaining areas of activity show basal cell carcinoma. To identify this growth clinically, the authors have called it "field-fire basal cell carcinoma." An important clinical point is that the growth may not appear serious; its diagnosis may even be missed, but in reality it is exceedingly malignant in that it may carry on by invasion primarily. When invasion has once become firmly established at the side of the nose, about the orbit or deep in the region of the ear, the growth is practically incurable. This point is important in prognosis and in teaching, as there has been a somewhat general idea that basal cell growths present little difficulty in treatment and cure. Moreover, the incidence of basal-squamous cell carcinoma of pure type may run as high as 20 per cent. Of all carcinomas, the basal cell type gives the best chance of cure by early removal, either by excision or by irradiation, but it is more dangerous to tissue and to life than is generally thought. H. A. MILLER, Philadelphia. [ARCH. OTOLARYNG.]

EXPERIMENTAL STUDIES WITH THE DERMATOPHYTES: IV. THE INFLUENCE OF AGE UPON THE ALLERGIC RESPONSE IN EXPERIMENTAL RINGWORM IN THE GUINEA PIG. EDWARD D. DELAMATER, J. Invest. Dermat. **5**:423 (Dec.) 1942.

The differences between the allergic reactions of guinea pigs of different ages during and after ringworm infection were studied. Three age groups were used: newborn animals, medium-aged animals and adult animals. Averages taken of the reactions of each group show that there is a development of the ability to react, from a low level in the newborn to a high level in the adults. Study of the variations met within each group shows that the ability to react allergically develops progressively with age. All newborn animals react in a typical manner with an allergy of low degree. The adult group was found to have acquired generally a higher degree of allergic reactivity. It is concluded that a maturation factor exists in the development of allergy to ringworm infections. The occurrence of the minimal type of allergic response in young animals is considered to offer a basis for explaining the observations that younger animals are more susceptible to progressive ringworm infections than older animals.

SWEAT RESPONSE TO ACETYLCHOLINE. DAVID KAHN and STEPHEN ROTHMAN, J. Invest. Dermat. **5**:431 (Dec.) 1942.

The local sweat response to intradermally injected acetylcholine (0.1 to 0.2 cc. of a 1:1,000 concentration of acetylcholine bromide in isotonic solution of sodium chloride) diminishes or disappears after sympathectomy. This observation is an exception to the rule of sensitization of structures after denervation. The local sweat response to the intradermal injection of acetylcholine is greater in men than in women.

SUNBURN AND PARA-AMINOBENZOIC ACID. STEPHEN ROTHMAN and JACK RUBIN, J. Invest. Dermat. **5**:445 (Dec.) 1942.

The comparison of the absorption spectrum of paraaminobenzoic acid with those of tryptophan and of proteins shows that, although the spectrums are similar, only paraaminobenzoic acid may be responsible for the shape of the sunburn action spectrum in the region of 3,000 angstrom units. Irradiated solutions of paraaminobenzoic acid cause inflam-

matory reactions if injected intradermally in man, whereas irradiated tyrosine, tryptophan, protein and nonirradiated solutions of paraaminobenzoic acid have no such effect. This observation supports the assumption, discussed as early as 1928, that ultraviolet erythema is due in part to the photochemical reaction of paraaminobenzoic acid in the skin. Paraaminobenzoic acid if incorporated into an ointment base protects against sunburn.

A Serologic (Nonsyphilitic) Reaction Approaching Universal Sensitivity. Reuben L. Kahn, Stanley Marcus, Elizabeth B. McDermott and Jacob Adler, J. Invest. Dermat. **5**:459 (Dec.) 1942.

The number of false positive reactions (nonspecific sensitivity) of a serodiagnostic test for syphilis can be readily increased by various modifications of the test. The use of excessively sensitive antigens may result in as high as 40 per cent of false positive reactions. The use of such antigens combined with the performance of the tests at a temperature of 1 C. may result in about 80 per cent of false positive reactions. If, in addition, unheated serums are employed (instead of serums heated for thirty minutes at 56 C.), the nonspecific sensitivity may approach 100 per cent. When a serodiagnostic test for syphilis is employed with animal serums (hog, chicken, horse, cow and rabbit) and the test is performed at a temperature of 1 C., close to 100 per cent of nonspecific sensitivity is obtained. These results suggest the existence of a universal serologic reaction which may afford a basis for detection of false positive reactions obtained with serodiagnostic tests.

Cutaneous Melanoblasts as Studied with the Paraffin-Dopa Technique. S. William Becker, J. Invest. Dermat. **5**:463 (Dec.) 1942.

The dopa reaction on 121 sections from cutaneous lesions has been studied with the paraffin section technic of Becker, Praver and Thatcher. The author is of the opinion that a lower percentage of positive reactions (64 per cent) was obtained than would have been obtained with the frozen section technic. The percentage of positive reactions was identical on exposed and on covered portions of the body; this would seem to eliminate the action of ultraviolet irradiation. The strongest reactions were found in sections from pigmentary and inflammatory dermatoses. Sections from a border of a vitiliginous patch demonstrated actual hyperpigmentation in this region. Sections from pigmented nevi and melanomas showed weaker reactions than are usually obtained with the frozen section technic. Cells showing transition between the clear cells of Masson and dendritic melanoblasts were seen, but no cells which could be considered transitional forms between palisade basal cells and melanoblasts were noted. This study lends weight to the concept that melanoblasts always arise as modifications of the clear cells. The paraffin section method of performing the dopa reaction permits of more exact cytologic study than does the frozen section method.

Male Hormone Stimulation Is a Prerequisite and Incitant in Common Baldness. James B. Hamilton, J. Invest. Dermat. **5**:473 (Dec.) 1942.

All men who failed to mature sexually had little or no dandruff and did not become bald. Common baldness occurs only in sexually mature persons of families the members of which tend to become bald. Androgenic therapy induced baldness in eunuchoid and castrate members of such families only. Cessation of treatment, as well as castration of normal men becoming bald, prevents further enlargement of bald areas but apparently does not promote general regrowth of hair. The author concludes that the predisposing factors, including congenital, are ineffective in production of seborrhea, acne vulgaris and premature alopecia in the absence of adequate gonadotropic substance. Androgens cause seborrhea, and acne may result. The presence of acne bears no direct relation to the levels of estrogenic and gonadotropic substances, and needs no postulation of hypopituitarism and hypogonadism. Premature alopecia is produced by daily intramuscular injections of 20 to 30 mg. of testosterone propionate, a dose estimated to provide androgenic stimulation roughly equivalent to that in normal men. There is no evidence that premature alopecia is caused by excessive quantities or unusual types of androgens.

Skin Manifestations of Meningococcal Infection. G. B. Mitchell-Heggs, Brit. J. Dermat. **54**:283 (Nov.) 1942.

The author divides the cutaneous manifestations of meningococcic infection into those that occur during the acute infection and those that occur during the chronic infection. The eruptions occurring in the acute fever are:

1. Small evanescent rose pink or red macules or papules occurring on the first or second day of the disease. Urticaria may occur.

2. Petechiae, occurring in the mouth, frequently encountered after a few days of the infection.

3. Purpura and ecchymoses.

4. Herpes labialis, generally appearing on the second or third day, and herpes zoster, usually on the fifth day.

5. Serum rashes, which occur on the ninth to the twenty-first day.

6. Eruptions due to sulfonamide compounds usually appearing on the eighth day of administration. They may be either macular and morbilliform or associated with a number of scattered small papules.

The cutaneous lesions in the chronic infection are distributed in much the same way as in the acute fever: on the extensor aspect of the limbs, on the back and at points of pressure. There is often a generalized patchy erythema, which may be a manifestation of the circulating toxin; it is frequently stippled with petechiae, which can be detected by diascopy and have been shown to be due to emboli. These areas vary in diameter from 1 to 3 cm. and may be unassociated with petechiae or swelling. Nodular lesions, which are usually tender and suggest erythema multiforme in some cases and erythema nodosum in others, may appear on the legs. Purpura may be present.

Mitchell-Heggs stresses the importance of a complement fixation test of the blood as a diagnostic measure, in view of the difficulty of blood culture in certain cases.

BLOOD CHOLESTEROL VALUES IN CASES OF GENERALIZED PRURITUS. ANNE E. SOMERFORD, Brit. J. Dermat. **55**:98 (April) 1943.

The blood cholesterol value was estimated for 43 patients with generalized pruritus whose blood sugar and urea values were normal. An abnormality was found in over 60 per cent of the cases, and it is suggested that hypercholesteremia or hypocholesteremia may be the cause of the pruritus.

BLUEFARB, Chicago.

A STUDY OF ERYTHROEDEMA POLYNEURITIS (PINK DISEASE). T. A. RATCLIFFE, J. Ment. Sc. **87**:545 (Oct.) 1941.

Ratcliffe reports 15 cases of pink disease, 6 of girls and 9 of boys. The author states that sex differences are probably of no etiologic significance. The age of onset varied from 3½ months to 1 year 5 months, and the mortality rate in his series was 13.3 per cent. It was impossible to trace a familial incidence. In many of the cases the onset of the disease appeared to be correlated with some other acute illness. Photophobia, anorexia, hypotonia and cutaneous rash were almost constant features, the rash being of less frequent occurrence than the others. Apart from hypotonia, neurologic symptoms were uncommon. In the patients personally examined by the author, the knee and biceps jerks were exceedingly difficult to elicit, but in no case were they absent. All of the bodily musculature was atonic, which may account for the sudden death from cardiac failure to which children with this disease are liable. Sensation was examined, so far as is possible in a baby, and there appeared to be diminution of sensibility for painful stimulation. The spinal fluid was examined in only 1 case and was found to be normal.

The psychologic state of the child is characterized by excessive irritability. For the most part the child lies curled up in the "knee-elbow" position, the face buried in the pillow. If disturbed, he will cry and withdraw from the examiner. Photophobia is usually noted. In a second type of the disease the child crawls about continually or rocks for hours or continuously performs acrobatic somersaults for a long time. In the rare type of the disease there is extreme lethargy, the child being miserable and irritable. This probably represents the advanced stage. No specific treatment for the disease is known, but particular attention is paid to dietetic measures.

It has been stated that the incidence of pink disease has been increasing in recent years, but this is probably more apparent than real. The question of etiology is still unanswered. One theory maintains the disease is due to a dietary deficiency, but the missing factor is unknown. It does not appear to be any of the known vitamins. A second theory postulates that the disease is caused by a specific organism or filtrable virus. No actual virus or organism has ever been isolated. Some attempts have been made to show that the disease is due to an endocrine disorder, but nothing definite is known about this factor. The prognosis should be guarded and the treatment directed toward combating the symptoms, particularly toward abolishing the irritability.

CUTANEOUS NAEVUS WITH BUPHTHALMOS AND EPILEPSY. REDVERS IRONSIDE and DENNIS HILL, J. Ment. Sc. **87**:631 (Oct.) 1941.

Ironside and Hill describe a case of Sturge's syndrome, so called because in 1879 Sturge gave the first complete account of a rare syndrome comprising the coexistence of naevus flammeus of the skin, glaucoma or buphthalmos and epilepsy.

The naevus flammeus appears characteristically over one side of the face within the distribution of the divisions of the trigeminal nerve. In the authors' case it extended over various segmental areas of the body. The buphthalmos or glaucoma occurs on the same side as the facial nevus. Within the cranial cavity two types of lesions have been described: (*a*) a venous cavernous angioma of the meninges and brain, sometimes penetrating as deep as the ventricular system, or (*b*) telangiectasis of the pia-arachnoid, with shrinkage and sclerosis of the underlying cortex and deposits of lime salts in the second and third cortical layers but not in the pial vessels. Weber added a fourth sign to the syndrome, the characteristic appearance of the roentgenograms of the skull.

The case described by the authors is that of a soldier with naevus flammeus and buphthalmos, who had repeated fits on the opposite side of the body while on duty with his unit. He was of low mentality but by no means mentally defective. Roentgenograms of his brain showed no calcification of the cerebral sulci. The contralateral fits which the patient described resembled migrainous attacks, since they were preceded by teichopsia. The loss of consciousness and residual transient paresis of the left arm and leg, however, render it probable that he suffered from a type of jacksonian epilepsy, though no fit was actually witnessed in the hospital. In this patient no heredofamilial incidence of the syndrome in whole or part could be ascertained. The article contains a good list of references on this unusual syndrome.

BRACELAND, Chicago. [ARCH. NEUROL. & PSYCHIAT.]

NICOTINIC ACID AND APHTHOUS STOMATITIS. J. DAMIANOVICH and R. RAVIZZOLI, Arch. argent. de pediat. **16**:21 (July) 1941.

The authors report on 15 patients with aphthous stomatitis, 11 of them children between the ages of 12 months and 6 years, who were treated with nicotinic acid given by the oral route. The dosage varied between 75 and 200 mg. in twenty-four hours and was divided in three or four doses. It was generally well tolerated. Only 1 patient showed marked capillary vasodilatation after a total dose of 75 mg.

The effect was generally prompt, and complete cure was usually accomplished within three days.

Gingivitis is more resistant to the treatment. With this condition additional treatment in the form of topical astringents usually had to be employed. Abundant vitatmin B complex was provided in the diet.

MULTIPLE XANTHOMATOSIS TUBEROSA. M. ACUNA and A. PUGLISI, Arch. argent. de pediat. **16**:328 (Oct.) 1941.

The authors report a case of multiple xanthomatosis tuberosa, an extremely rare condition, in a boy 9 years old. Since the age of 5 years the child had had disseminated tumor masses distributed symmetrically around the neck, the upper part of both arms, the anterior and posterior surfaces of the thighs and both buttocks. The masses varied in size and were reddish yellow.

The liver was much enlarged. The tumor masses were of the type of lipoid granulomas such as are found in the Hand-Schüller-Christian syndrome.

There were no osseous changes. Various forms of treatment, including ultraviolet and roentgen therapy, were without effect.

SULFATHIAZOLE IN THE TREATMENT OF PYODERMA IN THE INFANT. A. M. LAVIN, Bol. Soc. cubana de pediat. **13**:239 (May) 1941.

The author reports 4 cases of severe pyoderma in infants. All of the patients were treated with sulfathiazole; 2 of them had pulmonary complications.

The therapeutic result was striking. The drug was given in a dose of 0.1 Gm. per kilogram of body weight; this led to a concentration in the blood of 4 to 8 mg. per hundred cubic centimeters.

The author recommends the use of the drug particularly for the disseminating forms of pyoderma but not for the localized forms with extensive development of furunculosis. He believes that for these forms the toxoid serum is preferable.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

Society Transactions

BROOKLYN DERMATOLOGICAL SOCIETY

MORRIS M. ESTRIN, M.D., *President*

SEYMOUR H. SILVERS, M.D., *Secretary*

Feb. 15, 1943

Kaposi's Sarcoma of Fifteen Years' Duration. Presented by DR. SEYMOUR H. SILVERS.

H. M., a Jewish man aged 70, complained of a rash on both feet of fifteen years' duration. He first sought medical advice three years ago because of swelling of the left leg. He received topical applications at that time. About a year ago he received two roentgen ray treatments at another clinic. Seven years ago he underwent a prostatectomy.

Examination shows edema of the left lower extremity involving the lower two thirds of the leg and the foot. The toes, foot and ankle show a diffuse dusky red eruption, and at the periphery are infiltrated papules and nodules. Extending upward on the left leg are isolated satellite, lentil-sized to pea-sized infiltrated dusky red papules and nodules. The right foot and toes are similarly involved, as well as the hands and fingers, but to a lesser degree. There is evidence of maceration between the toes and dystrophy of the toe nails.

DISCUSSION

DR. ARTHUR W. GRACE: I noticed that in this patient, as well as in the other seen tonight, there was considerable solid edema. What mechanism causes its production?

DR. JACOB SKEER: I wonder also whether or not the fibrosis or the infiltration of the Kaposi sarcoma had involved or impinged on the lymphatics to cause the lymphedema. Is it coincidental, or does the sarcoma have something to do with the edema? Usually edema appears at the ankles or feet when the sarcoma is on the feet. The foot swells; a solid edema of the ankle then develops, and finally elephantiasis may result.

DR. LOUIS J. FRANK: From my observations, I can state that most patients with this disease have edema of the feet, but I cannot explain the mechanism.

DR. ABRAHAM WALZER: Kaposi's sarcoma involves endothelial cells, and these cells include those of the lymph and blood vessels. The cells characteristic of Kaposi's sarcoma are probably developments of these. With involvement of many of these cells, the circulation is involved and edema of the part is induced.

DR. SEYMOUR H. SILVERS: I do not think that edema was present when the disease started in the few patients that I have seen. As a matter of fact, in 1 that I have followed for six years, edema is not yet present. I should say that edema is one of the end results of this disease, a result of obstruction such as may occur in any disease. Some persons are affected by the disease more than others, and the obstruction and consequent edema are more severe.

Kaposi's Sarcoma. Presented by DR. MORRIS M. ESTRIN.

J. R., an Italian man aged 68, complained of swelling of the left leg for the past thirty-five years. For the first ten years of this period the leg was painful, and for the subsequent twenty-five years his leg was without symptoms except the chronic edema. About two years ago nodules began to develop on the sides of the left foot, which have slowly progressed to the present status, involving the entire left leg. He received six roentgen ray treatments about a year ago, which resulted in regression of many of the lesions. Since then no new lesions have formed.

Examination shows an edematous swollen left leg with many nodules and pedunculated tumors and several scars. The color varies from a deep hemorrhagic red to a deep blue. There are a number of verrucous lesions involving the heel, sides of the left foot and tissues about the toe nails. There is one pedunculated lesion on the right foot, and a few lesions are present on the right leg.

DISCUSSION

DR. JACOB SKEER: This case impressed me as being unusual because of the eruption-like spread of the nodules, especially on one leg, and the peculiar nodules involving the toes.

True, many of the nodules are the dark red, angiomatous type seen with Kaposi's sarcoma, but I failed to notice plaques. The eruption appears to me to be one of the metastatic type such as one would see with fibrosarcoma. It is not uncommon for lesions diagnosed as Kaposi's sarcoma to turn out to be true sarcoma.

DR. HARRY LEAVITT (by invitation): I want to mention two points brought out in the discussion. First, does the slide show any angiomatous changes? There is no pigmentation and no extravasation of blood, as seen in Kaposi's sarcoma. The second question concerns the unilateral lesion. Is it possible to rule out Kaposi's sarcoma because the lesion is unilateral?

DR. DAVID M. DAVIDSON: I agree with the last two speakers. I believe this is Kaposi's sarcoma. The small individual lesions, the plaque on the side of the right foot and the separate lesion simulating angiofibroma seen on the left leg are often seen with Kaposi's sarcoma. The unusual feature is that there are so many lesions scattered over the leg. The histologic structure gives the impression of fibrosarcoma, but I have seen many such sections from Kaposi sarcomas. I was disappointed not to find any extravasation of blood in the slide, but possibly other sections taken from other sites will show this condition. I am inclined to believe that this is an unusual case of Kaposi's sarcoma.

DR. E. ALMORE GAUVAIN: I saw this patient several weeks ago. As nearly as I can recall, there were certain elements suggestive of Kaposi's sarcoma but the lesions were unusual and involved only one leg, without the amount of hypertrophy usually seen in a patient with Kaposi's sarcoma. I am not willing to accept this diagnosis.

DR. SEYMOUR H. SILVERS: I believe this is Kaposi's sarcoma. I cannot see anything in the histologic section to rule out the diagnosis. A careful examination of the slide shows proliferation along the walls of the blood vessels. This observation, together with the clinical picture, should substantiate the diagnosis of Kaposi's sarcoma. The pedunculated lesions are not unusual, nor is the history of thirty-five years' duration.

A Case for Diagnosis (Atrophy of Tissues and Pigmentation?). Presented by Dr. JACOB SKEER.

S. R., a 16 year old white boy born in the city of New York came to the dispensary of the Israel Zion Hospital complaining of brown blotches on the neck and back of four years' duration. He stated that he had always been obese and had received numerous injections of chorionic gonadotropin and other medicaments. He also received treatment for hay fever and grip. His mother frequently gave him "ex-lax."

The condition began on the back four years ago, and the patch on the left side of the neck is of three years' duration.

Examination discloses an irregular, oval patch of brown pigmentation on the left side of the neck with areas of atrophy in the center. In the right lumbar region, at the level of the first lumbar vertebra, there is a patch of pigmentation the size of a small orange with a concavity in the center due to loss of tissues. There are linear, mottled areas of pigmentation on the back along the belt line.

A complete blood count and examination of the urine showed nothing abnormal.

The histologic examination of the section of skin showed the following conditions: The squamous epithelium of the surface was laid in moderate papillary folds. These were covered by cornified material. The basal layer contained brown pigment. In some places the pigment extended into the stratum spinosum. A few round cells were seen in the papillary layer. There was a moderate infiltration made up mainly of lymphocytes and plasma cells around the blood vessels in the corium. The sebaceous glands and the follicles had practically disappeared. The sweat glands were also diminished in number. An occasional smooth, atrophic muscle fiber was seen. Elastic fibers were frequently broken up. The subcutaneous fat tissue was somewhat atrophic and showed moderate invasion by small round cells.

The sections from the neck showed a richer round cell infiltration in the pars papillaris. The corium appeared to contain more cellular elements than that in the sections from the back. Sweat glands were also more numerous. Otherwise, both sections showed essentially the same features.

The diagnosis was atrophy of the skin.

DISCUSSION

DR. ABRAHAM WALZER: All lesions appear to be nevi. The peculiar formation of these lesions indicates a congenital defect.

DR. ARTHUR W. GRACE: The lesions on the neck are pigmented but not atrophic. I doubt that phenolphthalein would cause this condition.

DR. C. THOMAS CHIARAMONTE: In view of the bizarre appearance of the lesions and their accessibility, I do not think one should overlook the probability of their having been self induced.

DR. DAVID M. DAVIDSON: The lesions on the right side of the face and neck appear to be in the early stages. There is an erythema with slight infiltration but no atrophy.

DR. JACOB SKEER: The parents state definitely that the lesion on the arm followed an injury two years ago. The first lesions were noticed on the back four or five years ago. Later the lesion on the neck was noticed. They can give no history of trauma to account for this lesion.

Herpes Zoster with Scarring and Alopecia in an Eight Year Old Boy. Presented by DR. LOUIS J. FRANK.

J. F., a Negro boy aged 8 years, first noticed blisters on the back of the right ear on Dec. 23, 1942. These spread rapidly to involve the chest, neck, shoulder, back and scalp. Associated with the onset of the eruption he had severe pain and headaches, which required hospitalization. There was no history of his having had or having been exposed to chickenpox. The therapy consisted of two courses of intravenous injections of sodium iodide in aqueous solution. Each course consisted of injection of 0.5 Gm. of the drug daily for three days. There were alleviation of the severe neuralgic pains and rapid resolution of the lesions.

Examination today shows an eruption of the right side of the neck, shoulder, chest, scapular region and ear and extending to the occipital region of the scalp, with a large area of baldness. On the aforementioned areas, there are irregular pea-sized to dime-sized areas of depigmentation, scarring and beginning keloid formation. The areas involved exactly cover the entire distribution of the skin supplied by the right cervical plexus.

DISCUSSION

DR. E. ALMORE GAUVAIN: As I recall, these lesions were of the gangrenous type. They were definitely depressed, with black incrustation.

DR. HARRY LEAVITT (by invitation): At the beginning of the patient's hospitalization, the lesions consisted of clusters of large vesicles, superficial in appearance. They burst spontaneously, oozing a large amount of serum. He had two or three episodes of high temperature and required sulfathiazole therapy. After a while, the vesicles took on a grayish green color. Some of them became almost black and appeared gangrenous. It was difficult to say whether this was a secondary infection.

DR. ABRAHAM WALZER: Herpes zoster seen in aged persons may appear as one big gangrenous mass. The fact that the individual lesion looks black or white does not necessarily imply that it is gangrenous.

DR. ARTHUR W. GRACE: I believe this is herpes zoster with infection and decided scarring and alopecia.

A Case for Diagnosis (Exfoliative Dermatitis Secondary to Psoriasis?). Presented by DR. NATHAN PENSKY.

G. H., a machine operator aged 57, had no eruption until two and a half years ago, when he had his first attack of pneumonia. He was given sulfapyridine, which brought rapid results. However, a dermatitis of the legs developed which was believed to be due to the drug. The eruption became generalized and remained somewhat as it appears tonight. In August 1942, the patient had chills, and the family physician diagnosed pneumonia of the lower lobe of the left lung. Sulfathiazole was given, with rapid response. The eruption disappeared suddenly, and the patient remained "completely cured" for four days, when a fine scale appeared on the body. The administration of sulfathiazole was continued, and the eruption again became generalized.

At the present time the patient presents a generalized erythroderma of the body and face, with scaling and pruritus. There is pronounced generalized adenopathy. Severe itching is present. The soles are thickened and show large patches of laminated scales, not unlike those of psoriasis.

The Wassermann reaction of the blood was negative. The smear of the blood was normal, except for showing 8 per cent eosinophils. A section of the skin showed a picture of chronic eczema and possibly of psoriasis.

DISCUSSION

DR. DAVID M. DAVIDSON: I agree with the first half of the diagnosis. I see no signs of psoriasis. The lesions on the soles do not simulate psoriasis of the soles, and the rest of

the body does not show any psoriasis. I think the latter part of the diagnosis should be eliminated.

DR. JACOB SKEER: I agree with the diagnosis of erythroderma with exfoliative dermatitis. There is no evidence of psoriasis.

DR. C. THOMAS CHIARAMONTE: This patient shows evidence of some maceration and has well demarcated plaques on the soles, following eczema, possibly mycotic in origin.

DR. NATHAN PENSKY: There is a question as to whether sulfathiazole caused the eruption. The patient had pneumonia two years ago and received sulfathiazole. He then had pneumonia a second time, and sulfathiazole was again administered. The eruption did not get worse, though use of sulfathiazole was continued. There is no evidence that the exfoliative dermatitis was due to the medication.

A biopsy showed the pathologic features of an exfoliative dermatitis. The epidermis showed features suggesting an underlying psoriasis. No evidence of a lymphoblastoma was seen. Scrapings from plaques on the soles did not contain fungi.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

FRANK C. COMBES, M.D., *Chairman*

PAUL GROSS, M.D., *Secretary*

March 2, 1943

Lichenoid Exudative Discoid Dermatitis. Presented by DR. A. BENSON CANNON.

J. E., a Jewish tailor aged 58, was admitted to City Hospital on June 25, 1942, with a history of a generalized eruption of two years' duration. His past history was irrelevant except for a generalized eruption including the mouth twenty-eight years previously. This lasted six weeks and followed his drinking copious quantities of beer. The eruption cleared after treatment with potassium permanganate baths, and the patient remained well until two years ago, when pruritic lesions developed on the hands; they soon spread to involve the entire body, giving the picture of a generalized erythroderma. Constant local therapy afforded no relief, and in May 1942 the patient went to Arizona, where he remained only two weeks because of the heat. He admitted that he had taken many laxatives.

Examination on his admission to the hospital, in June 1942, revealed an acute exudative erythroderma over the entire body. In areas on the trunk there were still nummular lesions which were discrete, slightly infiltrated and brownish red. The skin was thickened throughout. The patient was in continuous misery because of the pruritus. Generalized adenopathy was present.

The Wassermann reaction of the blood was negative. Results of urinalysis and blood counts were normal.

Histologic examination of a specimen of skin from the left side of the chest revealed a moderately edematous epidermis. The papillary layer of the corium showed foci of infiltration; in some areas these were nodular, and in others the infiltration was rather diffuse. These areas lay just beneath the epidermis and were formed of fibroblastic stroma containing lymphocytes, plasma cells and occasional large cells with multiple nuclei. The diagnosis was possible mycosis fungoides.

During the first two months of the patient's stay in the hospital his disease was marked by stubborn resistance to all forms of therapy. In spite of roentgen therapy and various local applications the pruritus and oozing persisted. Only rarely did he have a remission for a few days, and even then it was slight.

On two occasions it was noticed that angioneurotic edema of the lips developed. One attack followed the ingestion of tomatoes, and the second occurred after the drinking of orange juice.

The pruritus was at times so extreme that the patient gradually became exhausted. A blood transfusion was given to build up his general condition, and he was given a high vitamin diet and liver. He has received starch baths, potassium permanganate baths, local applications of paste of bismuth N. F. and other medicaments.

During the last two months the patient's condition has improved greatly. The lesions are no longer wet, and there are few new lesions. However, there are pigmentation and some thickening, and some discoid lesions on the trunk are still seen.

DISCUSSION

DR. ADOLPH ROSTENBERG: I believe the case fits into the group of cases reported by Sulzberger and Garbe as instances of lichenoid exudative discoid dermatitis. The disease at present is in the lichenoid phase, but in a few months it may show an entirely different picture. However, it may be only a chronic eczema, though the chronicity and the clinical picture are characteristic of the disease mentioned.

DR. A. BENSON CANNON: I thought it would be interesting to present a typical example of chronic lichenoid discoid exudative dermatitis due to sensitivity to an external allergen. It has already been established that the patient was highly sensitive to pyrethrum used as a spray and also to clorox. He had a severe attack within an hour after coming in direct contact with the aforementioned preparations. I have 37 similar cases in which one or more local substances were responsible for the eruption, and a cure followed when the patient's environment was changed. I have sent patients to all parts of the country, from places in the state of New York to New Mexico, Arizona, California and Florida. Should the patient not be free from his eruption two weeks after his arrival at a given place, I advocate a change to another climate. Exposure to sunlight, use of a bland oil or cream and ingestion of a high vitamin diet are the remedies that may help to clear the skin.

Scleroderma, Progressive Type. Presented by DR. A. BENSON CANNON.

W. D., a man aged 64, was admitted to City Hospital Jan. 9, 1943, complaining of stiffness of the face of twenty years' duration and stiffness of the hands of six years' duration. He stated that a local physician, called because of some acute illness, made a diagnosis of scleroderma twenty years ago. The patient feels that the condition of his face has remained the same during the entire period. Six months ago he noticed increasing stiffness and pain in the tips of his fingers, and this has progressed steadily. He has not been able to work for the past five years. The general health is good.

Examination shows the skin of the fingers to be hard, tight and shrunken, causing about 50 per cent limitation of motion. There are small superficial ulcerations over the knuckles. The nails are curled over the finger tips, and there are some redness and swelling about the nail beds. The face appears pinched, with deep folds in the lips. The skin is hard and not freely movable on palpation. Hyperpigmentation is present on the face, with loss of pigmentation around the hair margin on the forehead. Similar changes are seen on the upper part of the chest and on the thighs. The toes are less involved than the fingers. Both legs show varicosities, with some ulceration about the ankles. A number of red telangiectatic macules the size of a split pea are seen on the face, palms and arms.

The Wassermann reaction of the blood was negative. A dextrose tolerance test, blood count and urinalysis gave normal results.

DISCUSSION

DR. EUGENE TRAUGOTT BERNSTEIN: This is an interesting case for the following reasons: The patient has symptoms of acrosclerosis, and at the same time symptoms of the sclerodactylic type of scleroderma are present. Since Sellei, in Budapest, made a distinction between acrosclerosis and the sclerodactylic form of scleroderma, it is interesting to observe a transitional form. In acrosclerosis there is shrinkage, involving the face and the hands and feet, with loss of tissue while the soft qualities of the skin are preserved, whereas in the sclerodactylic form of scleroderma there is an opposite process, of inflammation, hardening, sclerosis and, as a consequence, mutilation. The present patient not only shows involvement of the mouth, which is getting smaller, but the phalanges have been lost and he has involvement on the chest with symptoms of scleroderma; hence I should say his disease is a mixed form of acrosclerosis and the sclerodactylic form of scleroderma.

DR. FRANK VERO: Sellei described as acrosclerosis the condition which formerly was generally called scleroderma with sclerodactylia. He used the name acrosclerosis to differentiate it from other types of scleroderma.

Lichen Planus et Atrophicus (Folliculitis Decalvans and Lichen Spinulosus—Little and McCafferty). Presented by DR. FRANK VERO.

F. T., a man aged 55, is presented from Vanderbilt Clinic with an eruption on the face, scalp, trunk and genitals of five months' duration.

On the left side of the face and chin, extending to the neck, there is an irregularly defined erythematous scaly eruption with a few small sharply defined slightly atrophic central areas. On the scalp are numerous dime-sized, slightly reddened and atrophic areas showing definite alopecia. There are large irregularly defined patches over the frontal areas consisting of pin-

point-sized follicular closely grouped lesions and keratotic plugs. On the trunk and arms are similar slightly raised follicular papular lesions ranging in size from that of the point to that of the head of a pin and closely grouped. On the hard palate and the buccal mucosa are numerous pea-sized white or grayish patches, some surrounded with an erythema. The patient presents the most typical lesion on the penis, in the form of solitary and grouped flat-topped polygonal papular lesions; there are also annular and oval lesions with an atrophic center. Some nails show linear striation.

Biopsy of a specimen from the scalp suggested lichen planus et acuminatus, while a specimen from the chest suggested rather perifolliculitis.

Orthostatic Lymphedema of Penis and Scrotum. Presented by DR. GERALD F. MACHACEK.

J. E., a Puerto Rican aged 47, was admitted to the City Hospital on Jan. 12, 1943. Five months previously he had first noticed a painless swelling of the penis. At the same time the scrotum became larger.

The patient states that he has not had syphilis and that he has had no extramarital sexual relations during the past eight years. His wife, who suffered injuries to her eyes about ten years ago, is said to be well. Eight years ago the patient had gonorrhea, for which he was treated for one year, but a blood test was not made at that time.

For about twenty-five years the patient had had spells of dizziness at irregular intervals. Occasionally he loses consciousness and falls to the ground; attendants at the hospital have witnessed such episodes. Catheterization of his right eustachian tube has given only slight relief from these attacks. Twenty-five years ago he injured his left testicle while horseback riding; the scrotum increased gradually in size until at the end of one year it was of enormous dimensions—approximately "the length of his forearm." The scrotum was incised at the Veterans' Bureau in Puerto Rico and a large amount of thin fluid evacuated. About nine years ago he was treated at Harlem Hospital for a swelling of a gland in the right inguinal region; the swelling was incised and a binder applied. Within a few months the patient was well, and he has had no further trouble with his right groin.

On his admission to City Hospital the scrotum was edematous; the testicles were normal in size and not tender. The prepuce was also edematous but could be retracted with ease.

The Wassermann reaction of the blood was negative, and results of a urinalysis and a blood count were not remarkable. A cell count performed on the spinal fluid disclosed 80 cells per cubic millimeter. A Frei test performed on January 13 gave a positive reaction.

The patient was put to bed, and a course of sulfathiazole was given. After three days the edema of the scrotum and prepuce disappeared completely, and the patient was permitted to get up and walk about the ward for several days. During this time the edema recurred. On two occasions while arising from his chair he fell to the floor. He stated that there was no warning signal; he suddenly became dizzy and then unconscious and fell to the floor. He evidently regained consciousness after several minutes.

Massive Angioma Cavernosum Treated with Solid Carbon Dioxide. Presented by DR. ANDREW H. MONTGOMERY.

B. A. P., a girl now aged 21 months, was brought to the department of dermatology and syphilology of the New York Polyclinic Medical School and Hospital on Aug. 19, 1941, at which time she was 2½ months old. She had had from birth an extensive cavernous angioma involving the area about her right shoulder and completely surrounding that arm down to the hand. The limb was enlarged to more than twice the size of the other arm by the lesion, which consisted of large convolutions and valleys of a bright red color with no normal areas of skin. There was a malodorous discharge from maceration and fissures in the depths between the folds. On the posterior medial surface of the arm there was a large deep-seated ulcer. The child was frail, anemic and underweight.

Attention was directed at first toward improving the child's general condition. Locally, dressings wet with 1:3,000 solution of potassium permanganate were prescribed in order to clean up the field. Twice weekly the open areas were swabbed with 1 per cent aqueous solution of either gentian violet (methylrosaniline) or brilliant green (tetraethyldiaminotriphenylmethane sulfate). With the idea of inducing spontaneous involution, an attempt was made to shrink the lobulated elevations by frequent applications of contractile collodion.

Active therapy was not begun until March 2, 1942, just one year ago. It was decided that injection therapy, the method of choice for more limited lesions, was unsuitable in this case. For the past year, the lesion has been treated with solid carbon dioxide at intervals of about two and one-half weeks, pencils 1 cm. in diameter being applied with pressure and for a length

of time just short of the production of sloughs. About ten areas were treated at each sitting. As the lesion regressed, smaller applicators were used.

At present the arm is of the same size as the other. It is covered with a thin flesh-colored elastic scar, with no keloidal formation, no contractures and no limitation of motion. There are several areas of normal skin. Over the arm and at the upper and lower borders there still remain scattered punctate hemorrhagic lesions and minute angiomas.

DISCUSSION

DR. CHARLES WOLF: I believe this is the most striking case that we have had the opportunity to observe tonight. It brings home a forceful lesson, for the kodachrome photograph of this child demonstrated as extensive an angioma of an extremity as one would ever see.

Dr. Montgomery is to be complimented on the results obtained. He is to be complimented, furthermore, because this type of treatment for such an extensive angioma is looked at askance. Radiotherapeutists are particularly disinclined to feel that solid carbon dioxide has a place in the treatment of extensive angiomas. They rely on radium applied interstitially or by surface application. After such a result as this has been seen, one can say that solid carbon dioxide is still a reliable therapeutic agent.

DR. ADOLPH ROSTENBERG: I, too, am one of the old timers who still believe in the efficacy of solid carbon dioxide. I have never given up its use, and I have rarely seen a failure from it. I have had an opportunity to compare the results with those obtained with radium, and solid carbon dioxide is every bit as efficacious. The thickness of the lesion and not its size determines the effectiveness of the treatment. I believe that Dr. Gottheil was one of the first to use it after Dr. Pusey recommended its use, and I think it is one of the most useful therapeutic means available for treatment of such angiomas.

Chronic Lymphatic Leukemia (Cutaneous Leukemid). Presented by DR. GERALD F. MACHACEK.

G. M., a white man aged 66, is presented from City Hospital with a generalized eruption of eight months' duration. On June 27 the patient injured the skin of his right thumb. Three days later at the site of the scratch there appeared itching vesicles. These rapidly spread to both forearms and arms and to his face. On June 30 he was admitted to the City Hospital with edema of both forearms and arms. The skin was red, hot and tender. There was lymphangitis on the flexor surface of the right arm. The skin of both forearms, especially the right, was covered with crusts occurring in large plaques. The temperature was 103 F. That evening an admission diagnosis of impetigo with cellulitis was entertained.

On the morning of July 1, the total white blood cell count was found to be 40,000, with lymphocytes 75 per cent, polymorphonuclear leukocytes 7 per cent and monocytes 3 per cent. The remaining cells counted were found to be largely degenerated smudge cells. The lymphocytes were mainly young lymphoblasts, as indicated by their peroxidase-negative staining character. The hemoglobin content was 80 per cent, and the red blood cell count was 4,500,000 per cubic millimeter.

The patient was given a three day course of sulfathiazole for the eruption on his hands and arms, which cleared up by the fourth day. On this day his temperature dropped to normal and remained so. From this time on the patient, who had previously been in apparently good health, began to grow weak and lose weight, going from 154 pounds (69.9 Kg.) just prior to admission to the hospital to 134 pounds (60.8 Kg.) within one month after admission. The total white blood cell count continued to rise, reaching 100,000 on July 21, with 85 per cent lymphocytes, most of which were in the lymphoblast stage. The spleen was never definitely palpable, but the glands in the neck, axillas and epitrochlear region and in the groins were definitely enlarged.

For about three weeks after his admission to the hospital there were constantly recurring crops of pruritic crusted lesions on the face and upper extremities. These were treated as they developed by local cleanliness and wet dressings. They would always yield relatively soon after such therapy, but never completely. Asthenia and anorexia increased.

On July 29, 1942 a mass developed on the right cheek, and within three days it became the size of a robin's egg. This was treated with 100 r of roentgen irradiaton on August 6, with no relief. The mass was hard and nonfluctuant, and biopsy showed decided spongiosis and acanthosis of the epidermis with extensive areas of fibrinoid degeneration in the papillary layer of the corium. Several small foci of infiltration by lymphocytes, plasma cells and moderate numbers of eosinophils were noted in the same layer.

On August 9, after the white blood cell count had risen to 124,000 with 90 per cent lymphocytes (largely immature) and 8 per cent granulocytes, the splenic area was irradiated through two portals, an anteroposterior and a lateral, giving a total depth dose of 75 r filtered through 3 mm. of aluminum with a voltage of 125 kilovolts. Thereafter the mass on the cheek underwent rapid involution. The patient's appetite and strength improved, and the crops of pruritic crusted lesions no longer appeared.

On September 4 the white blood cell count rose to 67,250 and irradiation was again given through two portals. The spleen this time received 40 r filtered through 3 mm. of aluminum with 125 kilovolts. The mass on the right cheek was reduced to one-third its size within three days.

On September 10 the total white blood cell count was 57,000, with lymphocytes 68 per cent, polymorphonuclear leukocytes 15 per cent, monocytes 2 per cent, eosinophils 3 per cent and basophils 3 per cent, with 3 per cent of other types. The patient was not gaining weight, felt asthenic and was not eating well.

On November 14 he was given "spray irradiation" with the following technic: He was laid on the floor. The anterior surface of the body was divided into two portals, above and below the xiphoid process. Each of these portals was given 17 r, at a distance of 90 cm., filtration through 0.25 mm. of copper and 1 mm. of aluminum, with voltage of 135 kilovolts. Three days later the same factors were used on the posterior surface of the body, the dividing line being the skin at the level of the first lumbar vertebra. From this time until Feb. 14, 1943, the patient regained weight. On February 15 his weight was 143 pounds (64.9 Kg.). The total white cell count had remained around 30,000, with the lymphocytes varying between 70 and 75 per cent.

On February 15 the total leukocyte count rose to 49,000, with 80 per cent lymphocytes, mostly young cells. The patient's appetite was somewhat lessened, and he had several crusted lesions on the chin. All the glands are now large again. The glands of the left inguinal region were given 200 r through 0.25 mm. of copper and 1 mm. of aluminum, at a distance of 40 cm. and with a voltage of 135 kilovolts.

Syphilitic Gumma of the Eye. Presented by DR. J. LOWRY MILLER.

E. S., a white woman aged 31, was admitted to City Hospital on Dec. 15, 1942, complaining of ulceration of the left eyelid, exophthalmos of the left eye and ulceration of the palate and the vocal cords. The patient was well until November 1942, at which time she noted a painless swelling of the left lower eyelid. Within two weeks the eyeball and the left upper eyelid became swollen. At the same time severe hoarseness with complete loss of voice occurred. She was hospitalized in a hospital for patients with diseases of the eyes from December 5 to December 15, when it was noted that she had ulcerative lesions of the left eyelids and eyeball, and of the throat and larynx. During her stay there she lost weight and became rather feeble. She coughed a great deal. A roentgenogram of the chest revealed two lesions in the lower lobe of the right lung and one in the central area of the left lung, which were suspected as being of possible tuberculous origin. Examinations of the sputum did not reveal organisms.

On her admission to City Hospital she gave a history of chronic cough for one year, productive of white but never of blood-stained sputum. She had lost 30 pounds (13.6 Kg.) in weight in the preceding six months. There had been no night sweats, but dyspnea on exertion and edema of the ankles had been present for five months. She had had no blood tests before the onset of the present illness and gave no history of any sign or symptom of secondary syphilis. She had had one child, who died at the age of 11 months (cause of its death unknown) ten years ago, and there were no other pregnancies. She has been separated from her husband for many years. Nine months before she had had an extramarital sexual contact.

Examination on her admission to the hospital showed an emaciated woman who could not speak above a slight whisper. There were definite exophthalmos and photophobia of the left eye. The left upper and lower lids were swollen and red, and the outer palpebral conjunctiva was ulcerated, with slough on a granulating base. In the region of the left medial rectus muscle there was another ulceration through to the sclera. At the base of the uvula and on the adjacent portion of the soft palate ulcerations were present. Both false vocal cords were so red and swollen as to obscure the true vocal cords.

The Wassermann reaction of the blood was 4 plus. A roentgenogram of the chest on Feb. 16, 1943 revealed nothing abnormal, and the sputum did not contain tubercle bacilli. The blood count showed moderate secondary anemia. The spinal fluid was normal.

Treatment was begun with 30 minims (1.85 cc.) of saturated solution of potassium iodide three times daily. After she had received 90 minims (5.5 cc.), the laryngeal lesion showed signs of improvement, as did the ulcerations in the pharynx. She has had four intramuscular injections of solution of bismuth sodium tartrate of 1 cc. each, followed by thirty injections of 0.06 Gm. of arsenoxide given daily. There has been striking improvement from this therapy. The patient now speaks audibly. The exophthalmos is reduced, and the pharyngeal ulcerations are healed. The eyelids are not yet healed. She has been gaining weight steadily.

MANHATTAN DERMATOLOGIC SOCIETY

FRED WISE, M.D., *Chairman*

ANTHONY C. CIPOLLARO, M.D., *Secretary*

March 9, 1943

Folliculitis Ulerythematosa Reticulata. Presented by DR. E. W. ABRAMOWITZ.

S. R., a woman aged 26, came to the New York Skin and Cancer Hospital on Feb. 10, 1943, with dermatitis venenata of one hand. In addition, she showed an eruption on the cheeks. These areas presented worm-eaten-like pitted scars situated on a mild erythematous base.

The patient states that the eruption on her face has been present as long as she can remember. A maternal aunt has a similar condition, but none of the patient's siblings, parents or other relatives are affected.

DISCUSSION

DR. GEORGE C. ANDREWS: I agree with the diagnosis. This is a case of mild but definite folliculitis ulerythematosa reticulata.

DR. PAUL E. BECHET: I agree with the diagnosis despite the fact that there is no erythema and that only the reticulated atrophy is present. In my experience such a clinical picture is not unusual in the end stage of the disease. Ulerythema represents the active stage, and as the redness subsides there is nothing left but the reticulated scarring.

DR. DAVID BLOOM: I agree with the diagnosis. This disease is frequently found in several members of the family. In this case the family history is uncertain, for a maternal aunt who is believed to have a similar eruption on her cheeks is abroad and it is impossible to verify the statement of the patient.

DR. GEORGE M. LEWIS: I agree that the features are those of the presenting diagnosis. I believe the condition is postinflammatory and not nevoid.

DR. FRED WISE: I am in agreement with the opinion that the patient has a relatively mild and undeveloped manifestation of folliculitis ulerythematosa reticulata.

DR. E. W. ABRAMOWITZ: Dr. MacKee saw this patient at the clinic and agreed with the diagnosis. It is a mild form of this dermatosis. The patient states that her cheeks are highly colored, even when she does not use rouge.

A Case for Diagnosis (Hypertrophic Gingivitis?). Presented by DR. DAVID BLOOM.

M. W., a woman aged 27, came to the New York Skin and Cancer Hospital on Jan. 18, 1943, complaining of a sore mouth of nine months' duration.

The upper and lower gums on the right side present considerable hypertrophy, the excessive tissue protruding between the teeth. The patient complains of discomfort, particularly when eating.

This abnormality started when the patient was in the fifth month of pregnancy and has persisted since then without any change. The patient said that she had not taken any drugs. She stated that until a few weeks ago she did not eat any vegetables and ate little fruit. Within the last few days similar hypertrophy has developed on the anterior aspect of the hard palate.

The Wassermann reaction of the blood was negative. The blood cell count and the differential count were about normal, except for a relative increase of lymphocytes and presence of 4 per cent polymorphonuclear eosinophils. Examination of the urine gave normal results. Bacteriologic examination of the discharge of the gums by smear and culture did not show

any Vincent bacilli, but there were found hemolytic streptococci, microaerophilic streptococci and other bacteria.

DISCUSSION

DR. JACK WOLF: I agree with the diagnosis. While the clinical picture is highly suggestive of Vincent's infection, the inability to demonstrate the organisms, the unilateral distribution and the relatively small amount of pain all militate against the diagnosis of Vincent's disease. I could see no evidence that the process is malignant, and I suggest that another biopsy be performed.

DR. MAURICE J. COSTELLO: It would be interesting to know whether the hypertrophy during pregnancy was more extensive than it is now. I advise good oral hygiene, including massage of the gums. I believe that this patient has hypertrophic gingivitis. It is my impression that hypertrophic gingivitis of pregnancy improves after delivery.

DR. GEORGE M. LEWIS: The fact that the organisms have not been found speaks against Vincent's angina, but the persistence of the condition after pregnancy is also against hypertrophic gingivitis. Because I believe Vincent's angina is still a possibility, I advise that neoarsphenamine be used locally or be given intravenously in test dosage.

DR. MAX SCHEER: Vincent's organisms are so easy to find and so common in most mouths that failure to find them is of value. I believe this disease to be hypertrophic gingivitis which has extended beyond its usual duration.

DR. WILBERT SACHS: I believe this to be hypertrophic gingivitis, which is not uncommon. However, it is not impossible that a patient with this disease may also be acquiring a prickle cell epithelioma. With regard to confusing the early development of prickle cell epithelioma with pseudoepitheliomatous changes, I believe such a discussion is irrelevant when one is considering lesions of a mucous membrane. The pearls and whorls found in pseudoepitheliomatous changes are those of cross sections of hair follicles and are obviously not present in the mucous membrane. In this case there were definite anaplasia and whorl formation. If another biopsy is performed, the tissue should be taken from the edge of the previous site.

DR. ISADORE ROSEN: I had an opportunity to show this patient to a group of oral surgeons, who are consulted about this disease more than dermatologists are. It was the consensus of these surgeons that hypertrophic gingivitis is not a common complication of pregnancy and that it is slow to respond to treatment of all kinds. They claimed that when it is severe improvement does not take place until the teeth in the involved area are removed. One of them reported that in a series of 300 pregnant women not 1 was seen with hypertrophic gingivitis.

DR. GEORGE C. ANDREWS: I believe there should be a second biopsy. In regard to treatment, the fact should not be lost sight of that cultural examination revealed hemolytic streptococci. I suggest that sulfadiazine be given internally and roentgen treatment be given through the cheek.

DR. ANTHONY C. CIPOLLARO: I agree with the diagnosis. Diphenylhydantoin sodium can cause hypertrophy of the gums, and there may be other drugs which can cause hypertrophy of the gingivae.

DR. FRED WISE: While I cannot detect any clinical evidence of malignancy, I advise that the lesion be kept under observation over a period of several months, to exclude more serious manifestations later on.

DR. WILLIAM ABRAMOWITZ: The most recent reference to hypertrophic gingivitis that I could find was a report of 2 cases by Dr. C. L. Schmitt, of Pittsburgh (ARCH. DERMAT. & SYPH. **40**:653, [Oct.] 1939). In both women the tumor occurred during pregnancy; in 1 of them, with each of 2 pregnancies. The patient presented tonight shows no evidence of Vincent's stomatitis. I suggest the use of estrogenic substance.

DR. DAVID BLOOM: I believe that this abnormality developed originally on the basis of factors produced by pregnancy. For some unknown reason it has persisted long after termination of pregnancy. It must be infrequent, for several textbooks dealing with lesions of the mouth which I have consulted do not mention hypertrophic gingivitis at all or do it only casually. In the differential diagnosis one must consider leukemia, in which a similar type of gingivitis may be the first manifestation of the disease. Although in this patient the only abnormality in the blood count is the relative lymphocytosis, repeated blood counts will have to be done in order to exclude definitely a lymphoblastoma. As to treatment, the suggestion of Dr. Andrew is well taken. Many years ago, the late Dr. Satenstein suggested roentgen treatment for pyorrhea of the gums, and I have observed its value in several cases.

Pseudoxanthoma Elasticum. Presented by DR. WILBERT SACHS.

G. V., a 53 year old white man, came to the New York Skin and Cancer Hospital for treatment of a verruca on the right thumb. During the examination erythrasma of the groin, alopecia of the scalp, eyebrows and body and numerous small lesions on the face and neck were found.

On the sides of the face and on the neck and postauricular region were discrete flesh-colored to yellowish round and oval papules about 5 to 8 mm. in diameter. There are similar lesions in the axillas and on the sides of the body.

The scalp shows a diffuse sparse alopecia. There are some hyperpigmentation and a rather mottled appearance.

Ocular examination showed no angioid streaks; urinalysis revealed albumin (2+). A blood count and a chemical examination of the blood gave values within normal limits. A medical examination showed hypertension and arteriosclerosis. Biopsy of a specimen removed from the lesion on the cheek showed pseudoxanthoma elasticum, and a specimen from the lesion on the scalp showed perifolliculitis.

DISCUSSION

DR. GEORGE M. LEWIS: The clinical features of pseudoxanthoma elasticum are lacking. The yellow color, coarse wrinkling and angioid streaks are not present. The age of the patient is also unusual for pseudoxanthoma. I believe senile elastosis would be a more logical diagnosis. The diffuse alopecia suggests some toxic cause.

DR. MAURICE J. COSTELLO: I agree with the diagnosis of pseudoxanthoma elasticum. The appearance of the skin in the areas usually affected by this disease is definite though indicating a mild condition. I believe that the diffuse alopecia of the scalp has been caused by pseudoxanthoma elasticum in the same manner as the alopecia of the bearded region and the axillas.

DR. DAVID BLOOM: Although the lesions on the neck and in the axillas are not typical, I am willing to accept the diagnosis as presented in view of the confirmatory pathologic observations.

DR. JACK WOLF: The histologic section shows evidence of pseudoxanthoma elasticum. The question is whether this unusual clinical picture can be explained on the basis of pseudoxanthoma elasticum or whether sections from the other parts—scalp, axillas and pubic region—would show a different histologic picture. The eruption is interesting enough to warrant the further study which is necessary before a positive diagnosis can be made.

DR. FRED WISE: The clinical appearance differs greatly from that in the cases of pseudoxanthoma elasticum which I have observed, but the histologic changes merit serious consideration.

DR. WILBERT SACHS: Under this light not all the lesions can be seen. There is alopecia of the scalp, neck and other areas of the body. Naturally we tried to associate the latter with the pseudoxanthoma lesions. Biopsy of a specimen from the scalp revealed a perifolliculitis, with no degeneration or loss of the elastic tissue. One must then assume that the two processes are fundamentally different. I believe that it is comparatively easy to differentiate senile elastosis and pseudoxanthoma elasticum. The pathologic picture in this case is certainly not that of senile elastosis. I am unable to correlate the alopecia and the pseudoxanthoma elasticum, and I have never before seen the two associated.

DR. GERALD F. MACHACEK (by invitation): Pseudoxanthoma elasticum usually shows a characteristic clumping of elastic tissue fibers in the deeper layers of the cutis.

A Case for Diagnosis (Sycosis Vulgaris or Lupus Erythematosus?). Presented by DR. HERMAN SHARLIT.

L. DeG., an Italian aged 64, came to the New York Skin and Cancer Hospital on May 25, 1942, with an eruption of both cheeks of one year's duration.

On the right cheek there is an oval-shaped ill defined plaque, which is erythematous and presents tiny pustules and a few follicular crusted lesions and telangiectasia. Hair is present, but it is sparse. On the left cheek in symmetric location, there is a plaque which consists only of telangiectasia.

On numerous occasions in the course of observation over several months, the plaque on the right cheek showed many follicular pustules and on other occasions no pustules at all.

The report of the biopsy was "lupus erythematosus (possibly syphilitic)." Examination of the urine gave normal results. The Wassermann reaction of the blood was negative.

DISCUSSION

DR. WILBERT SACHS: I do not believe one could readily mistake lupus erythematosus for sycosis vulgaris. In the tissue submitted there were no pustules or any other features of sycosis vulgaris.

DR. MAX SCHEER: The lesions on the upper lip may be traumatic. The patient said that he cut himself while shaving; crusting followed, and he picked at the scab. I believe the diagnosis to be sycosis vulgaris.

DR. DAVID BLOOM: Frequent observation of the patient has convinced me that this man has sycosis vulgaris or a mild type of lupoid sycosis. I have observed frequently, in addition to the lesion on the upper lip, numerous follicular pustules in the plaque on the right cheek.

DR. JACK WOLF: I favor the diagnosis of lupus erythematosus. The lesion on the upper lip is probably not part of the clinical picture. It is a recently acquired fissure resulting from a cold, surrounded by an inflammatory zone.

DR. PAUL E. BECHET: The lesion at the bottom of the nose is due to "picking." The lesions on the cheeks are symmetrically distributed, infiltrated, sharply outlined and violaceous. There are few if any pustules either on the lesions or on the adjacent hairy parts. The ears are involved. For these reasons I consider the diagnosis of lupus erythematosus decidedly more plausible than that of sycosis.

DR. ANTHONY C. CIPOLLARO: The clinical features and the histologic picture fit in with the diagnosis of lupus erythematosus.

DR. FRED WISE: I find it difficult to interpret the various lesions, and I believe that a considerable amount of investigation would be required to determine the precise pathologic changes on the scalp, face and ears. I can see no evidence of lupus erythematosus at the present time, there being an absence of epidermal manifestations on the cheeks and ears.

Dermatitis Venenata (Dermatitis Due to Shorts). Presented by DR. MAURICE J. COSTELLO.

M. R., a man aged 38, from the dermatologic clinic of Bellevue Hospital, states that he was given three pairs of shorts about two weeks ago by the home relief agency. He wore these garments without washing them. He is wearing the third pair at this presentation. He states that forty-eight hours after wearing the first pair he experienced intense pruritus of the penis and scrotum, which was soon followed by an intense redness and edema of these parts. Within the next few days the eruption spread to the bathing trunk area, and more recently it has become generalized, being folliculopapular. There are also moderate edema of the eyelids and circumorbital redness. The patient has a temperature of 100.5 F. and has symptoms suggestive of constitutional reaction.

NOTE.—This patient is now hospitalized for a generalized exfoliative dermatitis developing as a result of the aforementioned irritant.

DISCUSSION

DR. GERALD F. MACHACEK (by invitation): This case appears to be a typical example of so-called shorts dermatitis.

DR. GEORGE M. LEWIS: This is a typical case of shorts dermatitis.

DR. JACK WOLF: The patient presents the picture of eczema due to contact in combination with a dermatitis due to the erythematourticarial component—that is, the toxic absorption factor—hematogenously distributed, which accounts for the widespread character of the eruption. The question arises as to whether the latter is due to the absorption of the contact agent or to some intermediate metabolic product developing in the course of the eczematous process.

DR. PAUL E. BECHET: I agree with the diagnosis. This is the most extensive eruption of the sort that I have ever seen.

DR. E. W. ABRAMOWITZ: This is a case of typical shorts dermatitis. Keil (ARCH. DERMAT. & SYPH. **47**:242, [Feb.] 1943) expressed the belief that an ester gum made from abeitic acid and a special type of alcohol is the cause.

DR. MAURICE J. COSTELLO: This patient presents an eruption similar to that of others with shorts dermatitis that I have observed. Intense pruritus of the penis and scrotum follows the wearing of shorts processed by a new resin fabric finish. Soon the scrotum becomes the size of a grapefruit owing to the intense edema, which extends to the penis, often causing angulation of this organ. This is occasionally followed by inability to urinate. Within the next few days a folliculopapular eruption appears on the shorts area; at times it becomes generalized and causes circumorbital edema. Occasionally toxic manifestations of a general nature appear, such as fever, nausea and articular pains.

FRED WISE, M.D., *Chairman*

ANTHONY C. CIPOLLARO, M.D., *Secretary*

April 13, 1943

Basal Cell Epithelioma of the Left Axilla and Left Arm. Presented by DR. JACK WOLF.

Miss L. H., a woman aged 52, gives a history of an eruption in the left axilla dating back almost twenty-five years. The eruption started with an insignificant spot which gradually spread until it involved the entire axilla. About four years ago several new spots appeared on the left arm. During this period the patient received medical attention for a short time. Apparently the nature of the disease was overlooked at that time.

On examination the patient presents a denuded, red, moist, granular, somewhat hollowed-out and irregular ulcerated area, occupying the entire axilla and measuring approximately 3¾ by 2 inches (9.5 by 5 cm.). The lesion is surrounded by a slightly raised, fine, cordlike border. The area extending beyond the lesion toward the chest is deeply indurated; that extending toward the arm is less so. The left arm and forearm are edematous and considerably larger than the normal right arm and forearm.

On the inner aspect of the arm at about the middle, within the circumference of a silver dollar, there are three lesions, each the size of a small pea, with a raised border and central ulceration. The skin between and surrounding these lesions is red and angry looking.

The Wassermann reaction of the blood was negative. Histologic examination of tissue removed from the edge of the axillary lesion confirmed the clinical diagnosis of basal cell epithelioma.

DISCUSSION

DR. GEORGE C. ANDREWS: My opinion is influenced by the case of 1 patient I had with a similar eruption. It was an unusual case because the eruption did not respond to treatment. Many operations had been performed, and finally the patient lost his shoulder by surgical operation and died. There were several biopsies; all showed basal cell epithelioma. I advise another biopsy in this case. The eruption may be a basosquamous cell lesion.

DR. ANTHONY C. CIPOLLARO: If this lesion is definitely a basal cell epithelioma, it should respond well to roentgen irradiation. I do not suggest surgical intervention in that location, because an extensive operation would be required and there may result severe lymphedema and partial loss of function of the arm. In my judgment, radiation therapy such as the patient is now receiving is good therapy, and I advise strongly its continuance.

DR. MAX JESSNER (by invitation): I regard this as basal cell epithelioma in an unusual location. It would be interesting to know whether the round lesions outside the main lesion are also basal cell epitheliomas.

DR. JACK WOLF: Because of the unusual location and the long duration, the clinical picture is not that usually encountered. However, the clinical diagnosis of basal cell epithelioma was confirmed by histologic examination of tissue removed from the edge of the lesion. Thus far, the patient has received 1,500 r given at a distance of 30 cm. with 100 kilovolts and 5 milliamperes, with an aluminum filter of 3 mm. While not now particularly elevated, the border was characteristic for basal cell epithelioma one week ago, before radiation therapy was instituted.

Chronic Ulcer of the Leg Caused by Microaerophilic Hemolytic Streptococci (Meleney). Presented by DR. GEORGE C. ANDREWS.

The lesion began on the left leg three months ago as a furuncle and later developed into an ulcer. It was treated with sulfathiazole, wet dressings, ammoniated mercury ointment, gentian violet and other remedies, without improvement.

The patient first consulted me on March 22, 1943, at which time there was a red, swollen area, 6 inches (15 cm.) in diameter, with a central deep necrotizing ulcer 2½ inches (6.3 cm.) in diameter, surrounded by four satellite ulcers each about 1 inch (2.5 cm.) in diameter. The Wassermann reaction was negative. From cultures of material taken from the surface of the ulcer in broth, made of meat extract, and Loeffler's blood serum, Staphylococcus aureus was obtained that showed slight hemolysis. These cultures showed growth only after forty-eight hours' incubation. Tissue taken from the lesion and placed in meat extract broth was incubated under aerobic and partially anaerobic conditions. Growth of Staph. aureus was again obtained. Blood agar was inoculated and incubated under aerobic and partially anae-

robic conditions (10 per cent carbon dioxide). Growth of the organism was enhanced and wider hemolysis produced when the material was incubated under 10 per cent carbon dioxide.

With treatment with zinc peroxide medicinal dressings and sulfadiazine internally, the lesion is rapidly healing. Blood counts at weekly intervals have been normal. A biopsy shows an intense polymorphonuclear infiltration, with necrosis of deeper vessels and tissue, particularly the fat.

DISCUSSION

DR. DAVID BLOOM: For patients with gangrenous pyoderma I am in favor of directing attention particularly to supportive treatment. One has to consider not only the presence of a particular micro-organism but the soil on which it grows. Many underlying conditions may weaken the resistance of the patient to the micro-organism, and they have to be treated. In cases in which no detectible condition is present which might be responsible for the lack of resistance, general supportive and stimulating treatment like administration of foreign protein may be of value. I believe that this patient has received sulfadiazine for several weeks and no striking effects have been produced. I therefore favor the discontinuance of the use of this drug.

DR. ANTHONY C. CIPOLLARO: Symptoms shown by this boy are similar to those of a patient with pyoderma gangrenosum whom I treated successfully with sulfathiazole by mouth and by local application in the form of an ointment.

DR. MAURICE J. COSTELLO: I should continue the therapy to which this patient has responded so favorably. Since zinc peroxide medicinal cream is specific for this type of infection, its use should be continued. I have had 3 or 4 patients suffering with this disease in the last year. They responded to this form of treatment. I cannot agree with what Dr. Bloom said about reactions to sulfonamide compounds. Many of the patients in our series of cases at Bellevue Hospital (Sulfonamide Therapy in Dermatology, *New York State J. Med.* 2317-2319 [Dec. 15] 1942) had no untoward reaction even when sulfathiazole was administered over a long period; others had severe reactions when only small amounts of the drugs had been administered.

DR. FRED WISE: I do not agree with the opinion that the ulcer was related directly to any metabolic disturbances. I advise that the treatment with zinc peroxide be resumed. Only local treatment is indicated for this healthy boy.

DR. GEORGE C. ANDREWS: A decade ago this type of lesion was called pyogenic gangrene, but Meleney separated this entity. It is contracted sometimes by young people in general good health. Treatment with zinc peroxide is specific. I have some gramicidin, but I prefer zinc peroxide. Treatment with gramicidin is frequently not successful and is extremely painful. This patient is comfortable with zinc peroxide.

A Case for Diagnosis (Pustular Psoriasis?). Presented by DR. GEORGE M. LEWIS.

Mrs. D. M., a widow aged 52, had no disease of the skin previous to one month ago. Since that time she has had repeated crops of pustular lesions affecting the palms and soles. Pruritus has been troublesome. The lesions are both discrete and confluent, are deep seated and are difficult to rupture. Physical examination and routine laboratory tests revealed no abnormalities. The tonsils were not considered to be enlarged or infected. The teeth were free of apical infection. During the past few days, several erythematous scaly lesions have developed over the extensor surfaces of the legs. These suggest psoriasis.

DISCUSSION

DR. M. B. PAROUNAGIAN: I agree with the diagnosis.

DR. GEORGE C. ANDREWS: I maintain that the lesions on the palms and soles are not psoriasis. The patches on the trunk may be, but there is no evidence that those on the palms and soles are psoriatic. The history is not that of psoriasis. No mention has been made of this woman's tonsils. She has large, purulent tonsils. Her teeth are rather bad; there are several dead teeth and caries. There has been no roentgen examination or dental repair. She requires a thorough examination. I do not know if she has had a blood count made. My suggestion is to remove the tonsils.

DR. MAURICE J. COSTELLO: I agree with Dr. Andrews. I saw this patient's tonsils; they are definitely infected. There are several lesions on the body which resemble those of psoriasis, but I do not think they are lesions of pustular psoriasis. The duration of the

eruption is only two or three months, and the pustules are much larger and more superficial than those seen in pustular psoriasis.

DR. DAVID BLOOM: Because of the obstinate resistance to therapy of pustular psoriasis, I believe that the procedure of removing infectious foci, if such are present, is rational. If foci are not detected, it is worth while to try to stimulate the defense mechanism by foreign protein therapy, like injections of boiled milk.

DR. MAX JESSNER (by invitation): It was interesting to hear the different opinions on pustular psoriasis. I doubt that the existence of a pustular bacterid as an analog to a tuberculid has been proved. To be able to prove it one needs, in addition to the blood culture of the microbe, a really specific cutaneous test for pyococci. That I believe has not yet been elaborated. I tried it with different vaccines and filtrates, but without success. I prefer to regard the pustular lesions in this case as pustular psoriasis. In biopsies of very early lesions of this kind I have seen striking intraepidermal "microabscesses."

DR. JACK WOLF: The controversy in regard to pustular psoriasis has been raging for a long time. This case illustrates well the difficulties encountered in making the diagnosis of that disease. Unless there are confirmatory lesions of psoriasis on other parts of the body or unless the removal of a focus of infection is followed by cure, it would appear more logical to refer to these eruptions on the soles and on the palms merely as recalcitrant vesicular eruptions of the soles and/or the palms.

DR. E. WILLIAM ABRAMOWITZ: My understanding of an id is that it is hematogenous and therefore usually appears as a shower from time to time and is not lasting, unless a destructive lesion develops, as occurs in some of the tuberculids. Removal of "infected tonsils" may improve the general health. According to Billings, the removal of a true focus prevents the invasion of tissue by additional organisms from the primary focus. It does not disturb the organisms already planted in another area. The concept of a pustular bacterid is difficult to prove; so is the validity of the theory and application of focal infection and systemic disease (Reiman, H. A., and Havens, W. O.: Focal Infection and Systemic Disease, *J. A. M. A.* **114**:1, [Jan. 6] 1940).

DR. GEORGE M. LEWIS: There is no evidence of any focus of infection in this patient. I am not in favor of removing tonsils without evidence of disease.

DR. FRED WISE: I think that the diagnosis of pustular psoriasis should be entertained, but it cannot be made definitely without long-continued observation.

DR. GEORGE M. LEWIS: In the new nomenclature such eruptions are referred to as pustular eruptions of the palms and soles. Local treatment effects a cure in many patients. It usually requires three or four months to obtain results. Many such patients have their tonsils removed, without any favorable effect on the skin.

A Case for Diagnosis (Erythema Multiforme? Dermatitis Factitia?). Presented by DR. GEORGE M. LEWIS.

S. G., a war correspondent aged 31, has had an eruption on the back of his neck for the past nine months. It developed while he was assigned to work with the United States Navy. Small pustules have been noticed in adjacent skin. Considerable local therapy was administered, including sulfathiazole and boric acid ointments. He first came under my care on Feb. 25, 1943. Since then he has had a course of superficial roentgen therapy. Locally, the part has been bandaged after the application of 2 per cent ammoniated mercury ointment. At present there are semicircular erythematous scaly and crusted lesions on the back of the neck. A few small pustules are also present.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: I favor the diagnosis of dermatitis factitia.

DR. JACK WOLF: From the data at hand I think that no definite clinical diagnosis can be made. Alteration of the clinical picture or the development of new lesions elsewhere ultimately may help in establishing a diagnosis.

DR. MAX JESSNER (by invitation): Clinically the bullous lesions may be impetigo. Against this diagnosis is the fact that follicles are rarely involved, and this man has folliculitis too.

DR. DAVID BLOOM: The eruption impresses me not as dermatitis factitia but as a localized erythema multiforme or as familial benign chronic pemphigus of the type described by Hailey and Hailey.

DR. MAURICE J. COSTELLO: I do not think that this patient is a malingerer. There is no reason why he should be. The hazardous life he follows he chose voluntarily. I believe that he has some type of persistent erythema multiforme or Hailey's disease. The neck is a common location for this disease.

DR. FRED WISE: I agree with the suggestion that the eruption is probably one of familial pemphigoid of the Hailey and Hailey type.

DR. GEORGE M. LEWIS: Dermatitis factitia is considered only as a faint possibility. The curious bizarre configuration cannot be explained readily, but the site of the eruption and the type of person concerned are against a self-inflicted disease.

Necrobiosis Lipoidica. Presented by DR. E. WILLIAM ABRAMOWITZ.

L. S., a woman aged 43, came to the New York Skin and Cancer Hospital on March 22, 1943, with an eruption on the legs of one year's duration. The shins presented a number of round, kidney-shaped and irregular plaques with a whitish blue center and an infiltrated brown border. On palpation, the lesions felt sclerotic. Most of the lesions showed telangiectasia. The patient complains only of moderate pruritus. The past and family histories are irrelevant. The Wassermann reaction of the blood was negative. The urine was normal. The blood count revealed a mild secondary anemia. The sugar content of the blood was 100 mg. per hundred cubic centimeters, and the cholesterol content was 255 mg. The biopsy confirmed the diagnosis of necrobiosis lipoidica.

DISCUSSION

DR. DAVID BLOOM: Because of the somewhat increased amount of cholesterol in the blood the patient may benefit possibly from a fat-poor diet. She states that since she has refrained from the use of sugar in her diet the itching of the legs has subsided. She will be sent to the vascular department for a study of her vascular system, as necrobiosis lipoidica may be due to some weakness in the vascular system.

DR. MAURICE J. COSTELLO: I agree with the diagnosis.

DR. JACK WOLF: I agree with the diagnosis.

Lichen Planus Linearis. Presented by DR. E. WILLIAM ABRAMOWITZ.

S. T., a Negro woman aged 35, came to the New York Skin and Cancer Hospital on March 31, 1943, with a lesion on the chin and neck of three months' duration. On the left side of the chin extending down to the neck, there is a ½ inch (1 cm.) wide band consisting of deeply pigmented crusted lesions. No oral lesions are present. The lesions itch. The patient was treated in 1939 for syphilis, but the Wassermann reaction of the blood is now negative. Biopsy of a lesion on the chin confirmed the diagnosis of lichen planus.

DISCUSSION

DR. ANTHONY C. CIPOLLARO: This is an unusual form of lichen planus. Many such aberrant forms simulate chronic contact dermatitis, lichen chronicus circumscriptus, psoriasis, leukoplakia, etc. Careful clinical observation will reveal in most cases the true nature of the disease. I recently saw 1 patient with lesions of lichen planus on the left side of the neck and chest which simulated a dermatitis caused by the wearing of a flower and which persisted for three years. At the time I saw her, typical lesions of lichen planus developed elsewhere. She promptly responded to treatment with injections of a bismuth preparation.

Dermatitis Exfoliativa Following Dermatitis Venenata Due to Shorts. Presented by DR. MAURICE J. COSTELLO.

P. G., a 54 year old Puerto Rican, was presented at the March 1943 meeting of this society. He is presented again this evening because he now has a universal pruritic exfoliating dermatitis accompanied by severe swelling and inflammation of the eyelids, the scrotum and the penis. He has latent syphilis but has received neither a bismuth preparation nor mapharsen in over six months. The Wassermann reaction of his blood was negative, and the Kahn reaction was 4 plus. No arsenic was found in the blood on chemical examination. Treatment consisted of injections of 5 per cent solution of dextrose, potassium permanganate baths, applications of boric acid ointment, sedation and other symptomatic therapy.

DISCUSSION

DR. FRED WISE: I believe that the patient had a blood-borne eruption which originated from the shorts dermatitis on the middle of the body, an eruption analogous to an id.

DR. MAURICE J. COSTELLO: I presented this patient again at this meeting because I believe that this is the first time a dermatitis exfoliativa following a shorts dermatitis has been reported. It differs little from the severe dermatitis exfoliativa following arsphenamine therapy.

A Case for Diagnosis (Microaerophilic Hemolytic Streptococcus Infection of the Axillas, Inguinal and Perianal Region?). Presented by DR. MAURICE J. COSTELLO.

J. B., a 45 year old Negro, was admitted to the dermatologic wards at Bellevue Hospital on March 19, 1943 because of painful draining multiple abscesses and sinuses in the axillas, in the inguinocrural and perianal regions and on the buttocks and thighs.

The infection began about eight years ago in the axillas and on the buttocks. Later the perianal region became involved. The patient has been hospitalized several times for surgical incision and drainage of the abscesses. When admitted to the hospital recently, he presented the following conditions: (1) healed acne vulgaris with postacne scarring; (2) healed folliculitis keloidalis; (3) extensive boggy fluctuant abscesses and sinuses affecting the buttocks and spreading in all directions. (Here and there were sinuses exuding a thin seropurulent and serosanguineous discharge. Associated with the main fluctuant masses were numerous perianal sinuses), and (4) boggy painful irregular abscesses and sinuses in both axillas, with multiple sinuses and cordlike structures traversing these lesions.

A blood count, urinalysis and roentgen examination of bones and lungs indicated normal conditions. Smears were negative for acid-fast bacilli. The Frei reaction, the reaction to tuberculin and intradermal reactions were negative. Ducrey bacilli were not found. The patient has shown remarkable improvement with chemotherapy.

DISCUSSION

DR. MAURICE J. COSTELLO: When I read my paper at the 1942 meeting of the American Medical Association in Atlantic City (Microaerophilic Hemolytic Streptococcus Infection Causing Destruction of the Nose, *J. A. M. A.* **121**:36-37 [Jan. 2] 1943), Dr. Brunsting, in the discussion, stated that he had observed a number of cases such as the one I presented tonight in which lesions were caused by microaerophilic hemolytic streptococci. Dr. Meleney has reported a number of similar cases. In a patient I observed recently suffering from this disease the fistulous tracts in the perianal region could be probed 4 or 5 inches (10 or 12 cm.). Drs. R. L. Sutton Jr. and Mark M. Marks recently reported a case of this disease, which they called acne conglobata and perianal pyoderma (*J. A. M. A.* **121**:1344-1347 [April 24] 1943).

MINNESOTA DERMATOLOGICAL SOCIETY

S. E. SWEITZER, M.D., *President*

H. A. CUMMING, M.D., *Secretary*

Minneapolis, May 7, 1943

Lichen Sclerosus et Atrophicus. Presented by DR. C. W. LAYMON, Minneapolis.

Mrs. A. N., a white woman aged 58, complains of lesions on her arms, chest and abdomen which have been present for the past several months.

She presents extensive sclerotic patches on the back, chest, abdomen and flexor surfaces of the forearms. Similar lesions are present in the groin and on the vulva. Individual white papules presenting follicular dilatation can be seen. There are numerous spiny lesions over the shoulders which impart a nutmeg grater feel to the skin.

DISCUSSION

DR. H. MONTGOMERY, Rochester: There is an extensive involvement of the skin. It would not be difficult to confuse this patient's disease clinically with morphea guttata, which, however, it is not.

Arsenical Keratoses; Arsenical Epitheliomas. Presented by DR. C. W. LAYMON, Minneapolis.

Mrs. M. D., a white woman aged 61, noticed lesions on her palms, soles and trunk about five years ago. She took arsenic drops for pernicious anemia from 1921 to 1925.

Examination shows multiple keratoses of the palms and soles as well as of the dorsa of the hands. There are numerous superficial epitheliomas on the trunk.

DISCUSSION

DR. C. W. LAYMON, Minneapolis: The patient was given solution of potassium arsenite for pernicious anemia about fifteen years ago. She continued the medication for five years. Within the past year keratoses of the palms and soles and at least thirty epitheliomas on various parts of the body have developed.

Raynaud's Disease and Generalized Progressive Scleroderma. Presented by DR. H. E. MICHELSON, Minneapolis.

K. T., a white man aged 53 years, has noticed numbness of his finger tips for twenty-five years. From the time of a prolonged exposure to the cold two and a half years ago, the finger tips and toes became painful after every subsequent exposure to the cold, after which they would blanch and then turn purple. The disease has become progressively worse. He has not been aware of any change in the skin of his fingers. He complains of nervousness, particularly during such attacks.

The history revealed that the patient had been treated for a cardiac condition for more than twenty years. He complained of attacks of pain in the chest and tachycardia. The diagnosis of hypertensive heart disease was recently made.

On examination the fingers distally are shiny and smooth and the skin is firm. Elasticity in the involved skin is lost. To a lesser extent the toes and cheeks are similarly involved.

DISCUSSION

DR. C. W. LAYMON, Minneapolis: The case presented an interesting though difficult diagnostic problem. Vasomotor phenomena, such as characteristic Raynaud's disease, were first noted, followed by a shrinking process of the hands and face. We thought that it was a case of mild diffuse scleroderma of the type described by Sellei and recently discussed by Dr. O'Leary as acrosclerosis.

DR. L. A. BRUNSTING, Rochester: I agree with the diagnosis. When the disease is limited to the face and extremities and when there are associated vasospastic phenomena, the prognosis is somewhat better than for diffuse scleroderma. The results from sympathectomy have been disappointing, and this procedure is recommended now only when there is a high degree of vasomotor sympathetic disturbance, as shown by changes of color with heat and cold, excessive sweating and discomfort.

Acrodermatitis Chronica Atrophicans. Presented by DR. S. E. SWEITZER, Minneapolis.

J. J., a white man aged 64, first came to the outpatient department of Minneapolis General Hospital in September 1935, at which time there were areas of redness with white scales on both elbows. Three months later itching lesions appeared on the hands and on both feet, especially on the lateral aspect, spreading upward to the knees.

Results of serologic studies for syphilis were negative. The family history is noncontributory, except that the patient's mother had some eruption on her legs.

Examination shows the lesions to be limited to circumscribed areas on the hands, forearms, elbows, lower part of the thighs, legs, ankles and feet. The lesions are dusky red. In most areas the skin is atrophic and semitransparent, with cigaret paper wrinkling. A few areas are lichenified, and several others are inflamed and somewhat indurated.

DISCUSSION

DR. H. MONTGOMERY, Rochester: I cannot fit the histologic changes with those usually seen in acrodermatitis chronica atrophicans. There are several specimens for biopsy, but none of the sections shows a border (*Grenzzone*) between the epidermis and the infiltrate in the cutis, and there is none of the atrophy of the cutis that one would expect to see in a case of acrodermatitis of this long duration. The epidermis shows keratotic plugging and acanthosis and adjacent areas of atrophy which are suggestive of lupus erythematosus. Clinically, however, the patient has no constitutional or systemic symptoms. The atrophic areas on the hands could represent a high degree of simple senile atrophy. It is difficult to explain the lichenified areas on the elbows and knees. I think this is a chronic inflammatory dermatitis of some type, clinically not lupus erythematosus and histologically not acrodermatitis chronica atrophicans.

Keratoderma Climactericum. Presented by DR. S. E. SWEITZER, Minneapolis.

Mrs. D. C., a white woman aged 64, first noted thickened soles associated with tenderness, itching, burning and fissure formation seven or eight years ago. She has become progressively worse. She knows of no history of a similar ailment in her family, and her children are not affected at this time. Serologic studies for syphilis gave negative results.

Daily doses of 1 mg. of diethylstilbestrol were prescribed, and much improvement followed within two weeks. Use of diethylstilbestrol was discontinued, and the skin of the feet became affected as previously. For the past two weeks the patient has been given diethylstilbestrol again, and improvement has taken place.

Examination shows mild hyperkeratosis of the soles. The hyperkeratotic layer ends abruptly at the sides of the feet.

DISCUSSION

DR. L. A. BRUNSTING, Rochester: This disease is a form of neurodermatitis and occasionally is difficult to distinguish from psoriasis. It occurs usually in persons who are middle aged and somewhat obese and in whom there is slight tendency toward hypertension. Occasionally men are affected.

DR. F. W. LYNCH, St. Paul: I think this eruption is a characteristic example of the disease described by Haxthausen under the title keratoderma climactericum. As in many cases of this disease, itching and burning have been rather severe and only the feet are involved. The response to estrogenic therapy is noteworthy. These eruptions seldom occur at the menopause but, rather, several years later, as in this case.

This type of keratodermatitis may frequently be confused with psoriasis, but I do not think there is any relationship with that disease. The two diseases can be differentiated microscopically and clinically and by the therapeutic response. There is a close relation between this disease and neurodermatitis.

Aleukemic Reticuloendotheliosis. Presented by DR. H. E. MICHELSON, Minneapolis.

H. S., a white man aged 28 years, first noted a small tumor on his chest five months ago. Later other similar lesions appeared on his skin, and one testicle became enlarged. After roentgen therapy the cutaneous lesions disappeared and the involved testicle regressed in size. It was excised two weeks after treatment. Subsequently many new tumors appeared, and the other testicle became enlarged. The lesions were painless. The patient's general health was not affected. There was no loss of weight.

On examination numerous round tumor masses varying in size from 2 mm. to 3 cm. in diameter are seen. Most of the lesions are bluish red. Some have the color of normal skin. All are painless. In general the lesions are distributed over the entire surface of the body. Only the palms and soles are clear. The lesions are more numerous on the chest and abdomen. There are few lesions on the upper part of the back. One is present on the conjunctiva of the right eye. Lesions are present on the scalp. Sharply defined pigmented atrophic scars are seen at the sites of roentgen treatment. Generalized lymphadenopathy is palpable. The remaining testicle is enlarged, firm and nodular. Otherwise there are no remarkable physical findings. The liver and spleen are not palpable.

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: I was amazed to learn from Dr. Downey that the testicle may be the original site for aleukemic tumefaction. He said that this was not unusual, but I have never encountered it before in my personal experience. Of course I cannot give the final word on the diagnosis for this patient, but my clinical judgment would be that the lesions will gradually assume the characteristics of sarcoma.

DR. H. MONTGOMERY, Rochester: From the clinical standpoint, this condition would fit into reticuloendotheliosis or a monocytic leukemia. In one of the sections of skin there were a fair number of grooved monocytes, but in another there were a good many cells suggestive of Sternberg or Dorothy Reed cells, such as are seen in Hodgkin's disease. The lymph node revealed many mitotic figures but no Dorothy Reed or Sternberg cells. This combination of observations would make me lean more toward the diagnosis of Hodgkin's type of lymphosarcoma than toward the diagnosis of monocytic leukemia. Cases like this one, however, often need prolonged observation and repeated biopsies and smears before the correct diagnosis is arrived at.

Parapsoriasis. Presented by Dr. S. E. Sweitzer, Minneapolis.

Mrs. A. K., a white woman aged 68, first noticed lesions on her body at the age of 16 years. These vanished and reappeared at intervals with topical applications and ultraviolet irradiation. For the two weeks prior to her last admission to the hospital, April 12, 1943, the burning sensation of the skin and the scaling had become more severe.

Examination shows a generalized though not universal scaling erythematous eruption of the palms and soles. The color of the lesions is dusky to coral red. Excoriations are present.

DISCUSSION

Dr. H. Montgomery, Rochester: This biopsy specimen is rather poorly prepared, and there is shriveling of the epidermal cells as well as of the cells in the infiltrate. Therefore one should not be misled into a diagnosis of mycosis fungoides, as the distortion in shape and size of the cells is obviously the result of poor fixation. Furthermore, the infiltrate is not sufficiently dense for mycosis fungoides, and the other changes are consistent with parapsoriasis en plaques. This patient has had the disease for a great many years and has had repeated biopsies, the early ones and also some of the more recent ones all revealing conditions consistent with a diagnosis of parapsoriasis. This case would typify what Burkhart and I emphasized in our recent observations, that parapsoriasis en plaques remains as such and seldom develops into mycosis fungoides or any of the other lymphoblastomas. Our observations support those of McCarthy and are contrary to those of Keil, who would maintain that in practically all cases parapsoriasis en plaques develops into mycosis fungoides.

Dermatitis Herpetiformis. Presented by Dr. S. E. Sweitzer, Minneapolis.

F. H., a white boy aged 7 years, first noted an itching burning eruption on the buttocks and scapular region and a few lesions scattered on the trunk and legs. At no time were there any vesicles. The lesions were discrete on a nonerythematous base, with secondary excoriation.

The personal and the family history were noncontributory. Serologic studies for syphilis gave negative results, and blood counts were normal.

The patient was admitted to the hospital, where topical applications and heavy ultraviolet irradiation were of little benefit. While he was in the hospital, mycosis of the feet and hands was noted and treated.

In the outpatient department 7½ grains (0.5 Gm.) of sulfapyridine four times a day was prescribed but brought no improvement. Autohemotherapy and injections of bismuth subsalicylate in oil were used in turn.

Examination shows grouped discrete crusted lesions over the scapular region and buttocks. Itching is present. Vesicles are not seen.

DISCUSSION

Dr. L. A. Brunsting, Rochester: When the administration of sulfapyridine is associated with gastrointestinal upset, the drug may be administered in the form of enseals. In almost every instance dermatitis herpetiformis responds to treatment with sulfapyridine, but the benefit lasts only as long as the drug is administered. Some patients are able to get along with small amounts, such as 0.5 Gm. two or three times a day; others require more. There have been no reactions of intolerance, but I take precautions to check the urine and the blood counts and to see that the intake of fluids by mouth is plentiful.

Dystrophy of the Nails. Presented by Dr. H. E. Michelson, Minneapolis.

Miss S. T., a white woman aged 21 years, first noted trouble with her nails three years ago. She stated that the nails fell off entirely after slight trauma. She also complained of a chronic cough after which a small amount of greenish sputum was raised. Both the trouble with her nails and the cough followed an episode of pneumonia three years ago.

Examination shows a chilblain type of circulation, cold hands and feet with excessive perspiration. The finger nails are thickened and darker than normal. No subungual keratoses are present. There is some transverse ridging of the nails but no pitting. The nails of the great toes are similarly affected.

Laboratory examinations showed no abnormalities.

DISCUSSION

Dr. C. W. Laymon, Minneapolis: The patient has partial onycholysis of some of the nails. Others are dark and thickened and fall off completely at the slightest trauma. A number of weeks ago several of my associates and I who saw her at the university were unable to make a definite diagnosis.

Epidermolysis Bullosa Hereditaria. Presented by DR. F. W. LYNCH, St. Paul.

J. V., aged 17 months, is said to have had red moist areas present on his heels at birth. Blisters were noted on the hands within the first week of life and have since been present most of the time on the hands and feet. There has been an occasional blister on the back. His general health has been good except for the presence of anemia, which has been treated.

The mother, aged 28 years, has had a similar eruption throughout her life and is also presented now because of large blisters on the hands which followed heavy work. She had four brothers and sisters without similar trouble, and she knows of no one in her family who has had such a disturbance. This child has no brothers or sisters.

On the child's hands and feet now are several large tense blisters, and numerous round red areas are present at the sites of former blisters.

DISCUSSION

DR. L. A. BRUNSTING, Rochester: The mother's symptoms became much milder at the time of puberty, and at present bullae develop only at sites of excessive trauma.

Contact Dermatitis. Presented by DR. S. E. SWEITZER, Minneapolis.

J. M., a 5 month old white girl, was admitted to Minneapolis General Hospital on April 22, 1943, with an eruption of three months' duration. The patient was in the custody of the Welfare Board and lived at the Maternity Hospital. She was never breast fed, but was fed a formula of one-half evaporated milk with 5 per cent dextrimaltose and 2 per cent calcium caseinate. She was normal until 2 months of age, when an "eczematous" rash developed. She was given liquid petrolatum baths, and boric ointment was used on the skin. Ascorbic acid and crystalline vitamin D_2 (drisdol) in propylene glycol were also given. Various solid foods were tried and eliminated.

An accurate family history of allergic diseases was not obtainable.

On her admission a diet of whole milk and the regular diet for a 6 month old infant was given for twenty-four hours, after which the lesions flared up. Then a diet of Mull-soy (a commercial preparation of soybean flour, oil and lecithin, with dextrose, sucrose, calcium phosphate, calcium carbonate and sodium chloride) was used for three days, during which time the baby lost 700 Gm. The next diet included skim milk boiled for twenty minutes and cream of barley. During the past week Sobee (a proprietary mixture of soybean flour, barley flour, olive oil, sodium chloride and calcium carbonate) was given. Local therapy included application of boric acid ointment until one week ago. Thereafter 1 per cent crude coal tar in paste of zinc oxide was used on the left leg for two days. Its use was then discontinued, and petrolatum was applied to the entire body.

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: I believe that the severe exfoliative dermatoses of childhood, such as Ritter's disease and Leiner's disease, can be ruled out in this case.

DR. S. E. SWEITZER, Minneapolis: Use of only one thing was not discontinued—preparations with grease in them. Various salves with a petrolatum base have been used. This may be the cause of the eruption.

NOTE.—Use of these preparations has since been discontinued, and the patient has improved rapidly.

News and Comment

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY

The next examination of the American Board of Dermatology and Syphilology will be held in Chicago on Friday and Saturday, June 9 and 10, 1944.

The written examination will be held in various large cities of the country on Monday, May 8, 1944.

Applications must be filed before April 1, 1944.

Please send all new applications to the Assistant Secretary, Dr. George M. Lewis, 121 East Sixtieth Street, New York.

At the examination held by the American Board of Dermatology and Syphilology in Philadelphia on Nov. 5 and 6, 1943, the following candidates were certified: Samuel M. Bluefarb, Chicago; Hans M. Buley, Champaign, Ill.; Henry S. Cambridge, Chicago; Wolfgang A. Casper, Surgeon (R), United States Public Health Service, New York; Capt. Earl R. Cockerell, Albilene, Texas; David M. Cohen, Chicago; Molleurus Couperus, Los Angeles; Francis J. Fagan, Troy, N. Y.; Major Hervey A. Foerster, Oklahoma City; James Q. Gant Jr., Surgeon (R), United States Public Health Service, Bethesda, Md.; Louis Goldstein, Philadelphia; Milton S. Hartman, Brooklyn; Capt. Otto B. Hitschmann, New York; Lieut. Herbert H. Holman, Detroit; Harold M. Johnson, Honolulu, Territory of Hawaii; Naomi M. Kanof, Washington, D. C.; Samuel M. Kaufman, New York; Lieut. Comdr. Samuel R. Kaufman, Wilkes-Barre, Pa.; Kurt Loewenthal, Brooklyn; Lieut. Salvatore J. Messina, Boston; George E. Morris, Boston; Lieut. Leslie Nicholas, Philadelphia; Capt. Norman M. O'Farrell, San Diego, Calif.; Benjamin P. Persky, Cleveland; Capt. Francis C. Prunty, Urbana, Ill.; Ernst M. Rosenbaum, New York; Joseph J. Russo, Albany, N. Y.; Capt. Walter F. Schwartz, Pasadena, Calif.; George B. Sexton, London, Ontario, Canada; Major Thomas H. Sternberg, Boston; Maurice J. Stone, Surgeon (R), United States Public Health Service, New York; James G. Thompson, Jackson, Miss.; Major Edmund N. Walsh, Whiting, Ind.; Lieut. Stephen L. Weisman, Paterson, N. J.; Malcolm A. Winkler, Providence, R. I., and Charles Wolf, New York.

Book Reviews

Formulary and Handbook, The Johns Hopkins Hospital. Edited by John C. Krantz Jr. Price, $2. Pp. 253. Baltimore: John D. Lucas Company, 1942.

This formulary and handbook was assembled under the direction of the associate medical board of the hospital and edited by John C. Krantz Jr. Its purpose is to familiarize the prescriber with various kinds of medicaments from the pharmacy and certain routine therapeutic procedures employed in the hospital.

Its table of contents consists of two main divisions: (1) "General Information" and (2) "Medicinal Agents." The section on general information is practical and of everyday use to the general practitioner and to the specialist. The chapter on the treatment of acute poisoning is brief and complete.

The chapter on the treatment of diseases of the skin, in section 2, covers twenty-four pages. It contains the conventional formulas for the treatment of the common cutaneous diseases. The common mistake of giving the formula for Whitfield's Ointment as 12 per cent benzoic acid and 6 per cent salicylic acid instead of 6 per cent and 3 per cent respectively is perpetuated.

Because of the frequent occurrence of dermatitis venenata following the application of ethylaminobenzoate ointment (benzocaine ointment), it would be better if it had been omitted from the formulary.

This well edited and clearly presented little formulary of therapeutically efficacious, simple and available medicaments should be in every physician's library.

Directory of Dermatologic Societies *

INTERNATIONAL

Tenth International Congress of Dermatology and Syphilology

Oliver S. Ormsby, President, 25 E. Washington St., Chicago.
Paul A. O'Leary, Secretary-General, 102-2d Ave. S. W., Rochester, Minn.
Place: New York. Time: Postponed indefinitely.

Pan American Medical Association, Section of Dermatology and Syphilology

J. J. Eller, President, 745–5th Ave., New York.
Austin W. Cheever, Secretary, 464 Beacon St., Boston.

FOREIGN

British Association of Dermatology and Syphilology (Canadian Branch)

B. Usher, President, 1538 Sherbrooke St. W., Montreal.
Frederick Kalz, Secretary-Treasurer, 1414 Drummond St., Montreal.

Royal Society of Medicine, Section of Dermatology

H. W. Barber, President, 7 Devonshire Pl., London, W. 1, England.
Louis Forman, Secretary, 7 Devonshire Pl., London, W. 1, England.

Sociedad Mexicana de Dermatología

Jorge Millán Gutiérrez, President, Colima No. 208, Mexico, Mexico.
Pedro Daniel Martínez, Secretary, Lucerna No. 48, Mexico, Mexico.

NATIONAL

American Medical Association, Scientific Assembly, Section on Dermatology and Syphilolgy

Clark W. Finnerud, Chairman, 55 E. Washington St., Chicago.
Nelson P. Anderson, Secretary, 2007 Wilshire Blvd., Los Angeles.

American Academy of Dermatology and Syphilology

George M. MacKee, President, 999–5th Ave., New York.
Earl D. Osborne, Secretary, 471 Delaware Ave., Buffalo, N. Y.

American Board of Dermatology and Syphilology

Howard Fox, President, 140 E. 54th St., New York.
C. Guy Lane, Secretary-Treasurer, 416 Marlborough St., Boston.

American Dermatological Association

Joseph Klauder, President, 1934 Spruce St., Philadelphia.
Harry R. Foerster, Secretary, 208 E. Wisconsin Ave., Milwaukee.

Society for Investigative Dermatology

Fred D. Weidman, President, 1930 Chestnut St., Philadelphia.
S. William Becker, Secretary, 8 S. Michigan Ave., Chicago.

SECTIONAL

Central States Dermatological Association

John E. Rauschkolb, Secretary-Treasurer, 1808 Republic Bldg., Cleveland.
Place: Cleveland. Time: Postponed indefinitely.

* Secretaries of dermatologic societies are requested to furnish the information necessary for the editor to make this list complete and to keep it up to date.

MISSISSIPPI VALLEY DERMATOLOGICAL SOCIETY

Lawrence C. Goldberg, Secretary-Treasurer, 623 Doctors Bldg., Cincinnati.
Place: Chicago.

NEW ENGLAND DERMATOLOGICAL SOCIETY

Jacob H. Swartz, President, 371 Commonwealth Ave., Boston.
Francis M. Thurmon, Secretary, 520 Commonwealth Ave., Boston.

NORTHERN NEW JERSEY DERMATOLOGICAL SOCIETY

John Kiley, President, 94 Park St., Montclair.
Henry Abel, Secretary, 339 Union Ave., Elizabeth.
Place: Academy of Medicine of Northern New Jersey, Newark. Time: Third Tuesday of March, April, October and December.

SOUTHEASTERN DERMATOLOGICAL ASSOCIATION

Howard Hailey, Chairman, 107 Doctors Bldg., Atlanta, Ga.
A. H. Lancaster, Secretary, P. O. Box 585, Knoxville, Tenn.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Dudley C. Smith, Chairman, University of Virginia Hospital, Charlottesville, Va.
J. Lamar Callaway, Secretary, Duke Hospital, Durham, N. C.

STATE

CALIFORNIA MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, DERMATOLOGY AND SYPHILOLOGY SECTION

Irving R. Bancroft, Chairman, 1052 W. 6th St., Los Angeles.
Chris Halloran, Secretary, 1052 W. 6th St., Los Angeles.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON DERMATOLOGY

Harry Bailey, Chairman, 242 Trumbull St., Hartford.
Louis O'Brasky, Secretary, 1172 Chapel St., New Haven.

FLORIDA SOCIETY OF DERMATOLOGY AND SYPHILOLOGY

Wiley M. Sams, Chairman, 25 S. E. 2d Ave., Miami.
Lauren M. Sompayrac, Secretary, 117 W. Duval St., Jacksonville.

LOUISIANA DERMATOLOGICAL SOCIETY

M. T. Van Studdiford, President, 912 Pere Marquette Bldg., New Orleans.
R. A. Oriol, Secretary-Treasurer, 921 Canal St., New Orleans.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON DERMATOLOGY AND SYPHILOLOGY

John G. Downing, President, 520 Commonwealth Ave., Boston.
G. Marshall Crawford, Secretary, 1101 Beacon St., Boston.

MEDICAL SOCIETY OF THE STATE OF NEW YORK, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Harry C. Saunders, Chairman, 159 W. 87th St., New York.
James W. Jordan, Secretary, 471 Delaware Ave., Buffalo.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA, SECTION ON DERMATOLOGY

Bernhard A. Goldmann, Chairman, 500 Penn Ave., Pittsburgh.
M. A. Pettler, Secretary, 1319–8th Ave., Beaver Falls.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Frank Menaugh, Chairman, Ford Hospital, Detroit.
Eugene A. Hand, Secretary, 801 Second National Bank Bldg., Saginaw.

Minnesota Dermatological Society

S. E. Sweitzer, President, 825 Nicollet Ave., Minneapolis.
Harry A. Cumming, Secretary-Treasurer 419 Medical Arts Bldg., Minneapolis.
Time: First Friday in January, April, July and October.

Oklahoma State Dermatological Society

John H. Lamb Jr., President, Medical Arts Bldg., Oklahoma City.
M. M. Wickham, Secretary, 716 W. Symmes St., Norman.

Texas Dermatological Society

J. Lewis Pipkin, President, 705 E. Houston St., San Antonio.
Duncan O. Poth, Secretary-Treasurer, 414 Navarro St., San Antonio.

LOCAL

Baltimore-Washington Dermatological Society

H. Hanford Hopkins, Chairman, 1123 St. Paul St., Baltimore.
Reuben Goodman, Secretary-Treasurer, 1746 K St. N. W., Washington, D. C.
Place: Alternate cities. Time: Third Thursday of each month.

Bronx Dermatological Society

David Bloom, President, 135 E. 50th St., New York.
Henry Silver, Secretary, 290 West End Ave., New York.
Time: Fourth Thursday of each month from October to May, inclusive.

Brooklyn Dermatological Society

T. Chiaramonte, President, 1 Nevins St., Brooklyn.
J. Schweig, Secretary, 47 E. 61st St., New York.
Time: Third Monday of each month except June, July, August and September.

Buffalo-Rochester Dermatological Society

James W. Jordon, President, 471 Delaware Ave., Buffalo.
Frank A. Dolce, Secretary-Treasurer, 468 Delaware Ave., Buffalo.

Central New York Dermatological Society

Albert Van Vranken, President, 1565 Union St., Schenectady.
Maxwell C. Snider, Secretary, 106 Oak St., Binghamton.

Chicago Dermatological Society

Michael H. Ebert, President, 25 E. Washington St., Chicago.
Marcus R. Caro, Secretary-Treasurer, 25 E. Washington St., Chicago.

Cincinnati Society of Dermatology and Syphilology

Harry L. Claasen, President, Provident Bank Bldg., Cincinnati.
Lawrence C. Goldberg, Secretary-Treasurer, 623 Doctors Bldg., Cincinnati.
Place: Cincinnati. Time: First Wednesday of each month, except July, August and September.

Cleveland Dermatological Society

J. R. Driver, President, 1422 Euclid Ave., Cleveland.
G. W. Binkley, Secretary, 10515 Carnegie Ave., Cleveland.
Time: Fourth Thursday of each month from September to May, inclusive.

Detroit Dermatological Society

Robert C. Jamieson, President, 1553 Woodward Ave., Detroit.
James R. Rogin, Secretary-Treasurer, 2306 Eaton Tower, Detroit.

Kansas City (Mo.) Dermatological Society

C. C. Dennie, President, 1103 Grand Ave., Kansas City.
Thomas B. Hall, Secretary, 902 Professional Bldg., Kansas City.

Los Angeles Dermatological Society

William H. Goeckerman, President, 727 W. 7th St., Los Angeles.
C. Russell Anderson, Secretary, 1930 Wilshire Blvd., Los Angeles.
Time: Second Tuesday of each month, October to May, inclusive.

Manhattan Dermatologic Society

Anthony C. Cipollaro, Chairman, 40 E. 61st St., New York.
Wilbert Sachs, Secretary, 57 W. 57th St., New York.

Metropolitan Dermatological Society

Laird Van Dyke, President, 206 E. 17th St., New York.
Royal M. Montgomery, Secretary, 57 W. 57th St., New York.

Montreal Dermatological Society

Paul Poirier, President, 456 Sherbrooke St. E., Montreal, Canada.
Alphonse Bernier, Secretary, 3995 St. Denis St., Montreal.

New York Academy of Medicine, Section of Dermatology and Syphilis

David Bloom, Chairman, 135 E. 50th St., New York.
George M. Lewis, Secretary, 121 E. 60th St., New York.

New York Dermatological Society

Edward R. Maloney, President, 136 E. 64th St., New York.
J. Gardner Hopkins, Secretary-Treasurer, 630 W. 168th St., New York.

Omaha Dermatological Society

Donald J. Wilson, President, 1113 Medical Arts Bldg., Omaha.
Leonard J. Owen, Secretary-Treasurer, 954 Stuart Bldg., Lincoln, Neb.

Philadelphia Dermatological Society

Carroll S. Wright, Chairman, 1402 Spruce St., Philadelphia.
Carmen C. Thomas, Secretary, 1930 Chestnut St., Philadelphia.
Time: Third Friday of September, November, January, March and May.

Pittsburgh Dermatological Society

M. F. Pettler, President, 1319-8th Ave., Beaver Falls, Pa.
Frederick Amshel, Secretary, 3401 Fifth Ave., Pittsburgh.
Time: Third Thursday of every month except July and August.

St. Louis Dermatological Society

Norman Tobias, President, Missouri Theatre Bldg., St. Louis.
Joseph Grindon Jr., Secretary-Treasurer, 323 Lister Bldg., St. Louis.
Place: Barnard Free Skin and Cancer Hospital. Time: 2 p. m., second Wednesday of each month.

San Francisco Dermatological Society

Arne E. Ingels, President, 490 Post St., San Francisco.
Ervin Epstein, Secretary-Treasurer, 1904 Franklin St., Oakland, Calif.
Time: Third Friday of February, April, September and November.

INDEX TO VOLUME 48

The asterisk (*) preceding the page number indicates an original article in the Archives. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Biographies, Book Reviews, Editorials, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, E, O and S, respectively.

Abnormalities and Deformities: See under names of diseases, organs and regions, as Atrophy; Hair, abnormalities; Nails; Pigmentation; Skin, abnormalities; etc.
Abramowitz, E. W.: Folliculitis; case for diagnosis, 565
  Folliculitis ulerythematosa reticulata, 673
  Lichen planus linearis, 680
  Lymphoblastoma resembling neurodermatitis, 437
  Mycosis fungoides of chest with parapsoriasis en plaques of body, 427
  Necrobiosis lipoidica, 680
  Persistent marginal blepharitis (due to yellow mercuric oxide), 443
Abscess: See under names of organs and regions
Acanthosis nigricans; case for diagnosis, 109
  nigricans; is it form of avitaminosis? *650
  nigricans of juvenile type, mild diabetes and obesity, 468
Acariasis, dermatitis due to Tyroglyphus longior, Gerv., var. Castellanii, Hirst, in cheese dust, 425
Acarus Scabiei: See Scabies
Acetarsone, Therapy: See Pemphigus
Acetylcholine: See Choline and Choline Derivatives
Achromia: See Vitiligo
Acid, Nicotinic: See Nicotinic Acid
  para-aminobenzoic, and sunburn, 661
  Salicylic: See Salicyl Compounds
Acne, extensive postacne keloids, 431
  halowax acne ("cable rash"); cutaneous eruption in marine electricians due to certain chlorinated naphthalenes and diphenyls, *251
  rosacea-like tuberculid of face, 555
  rosacea-like tuberculid of Lewandowsky, 333, 463
  urticata polycythemica accompanied by polycythaemia vera and loss of directional power in fingers of left hand, 430
  vulgaris, fat metabolism in, 207
  vulgaris with comedos; treatment by monoterminal electrodesiccation, *302
Acrodermatitis chronica atrophicans, 682
  chronica atrophicans with fibrous nodules or mycosis fungoides, 126
Acrodynia, erythredema polyneuritis (pink disease), 663
Acrosclerosis: See Scleroderma
Actinodermatitis: See under Light
Actinomycosis or neurotic excoriations, 550, 562
Adenoma sebaceum treated by solid carbon dioxide, *198
Adipose Tissue: See Fat; Lipoma; Obesity
Adolescence, variations in cutaneous manifestations of vitamin A deficiency from infancy to puberty, *1
Age, experimental studies with dermatophytes; influence of age on allergic response in experimental ringworm in guinea pig, 661
  Old: See Old Age
  variations in cutaneous manifestations of vitamin A deficiency from infancy to puberty, *1
Albinism: See Vitiligo
Allergy: See Anaphylaxis and Allergy
Alopecia, areata, *581
  depression-hyposexual-alopecia syndrome, 317
  herpes zoster with scarring and alopecia in 8 year old boy, 667
  male hormone stimulation is prerequisite and incitant in common baldness, 662
  traumatic marginal alopecia due to traction on hair; comparative study of alopecia liminaris frontalis of Sabouraud, *527
Amebiasis cutis, 571
American Medical Association, Section on Dermatology and Syphilology, 536
Amines, acquired hypersensitivity to chlorinated ethylamine vesicants; report of case, *15
Amyloidosis, congo red test for amyloid disease; quantitative technic; utility of congo red test in diagnosis and in differential diagnosis, 660
  cutis, 224
  cutis, localized, 112, 131
  of skin, 337
Anaphylaxis and Allergy: See also Asthma; etc.
  allergenic properties of vegetable gums; case of asthma due to tragacanth, 423
  cutaneous tests with hen's egg white fractions in atopic infantile eczema, *258
  experimental studies with dermatophytes; influence of age on allergic response in experimental ringworm in guinea pig, 661
  histologic studies of reactions to intracutaneous tests in allergy of infection in humans, 541
  newer interpretation of positive skin reactions, 98
  results of questionnaire on patch tests; analysis of answers by 211 dermatologists and 55 industrial physicians, *514
  studies in abnormal human sensitivity to light; passive transfer of light hypersensitivity in prurigo aestivalis, 543
  studies in abnormal human sensitivity to light; photoallergic concept of prurigo aestivalis, 543
Ancylostoma: See Hookworm Infection
Anderson, N. P.: Pagetoid epithelioma (scrotum); arsenical keratoses (palms and soles), 319
  Parapsoriasis; case for diagnosis, 320
  Piedra, 473
  Xanthoma; case for diagnosis, 471
Andrews, G. C.: Arsenical pigmentation and hyperkeratosis; case for diagnosis, 547
  Chronic ulcer of leg caused by microaerophilic hemolytic streptococci (Meleney), 677
Androgens, effect on sebaceous glands of human skin, *601
  male hormone stimulation is prerequisite and incitant in common baldness, 662
  mycosis fungoides with bullous lesions; report of case resistant to roentgen and arsenical therapy; effects of empiric therapy, partly based on laboratory investigations, *359
Anemia, sickle cell, in white race, 422
  sickle cell, with ulcerations, 558
Angioid Streaks: See Retina, blood supply
Angioma cavernosum, massive, treated with solid carbon dioxide, 670
  of head, 561
  Parkes Weber syndrome; hemangiectatic hypertrophy, 449
Ankle: See also Foot
  neurogenic arthropathy of left knee and ankle; gumma of left ankle; juxta-articular nodules due to syphilis; atrophic arthritis, 116
Anthrax, 421, *582
Antigens and Antibodies: See Lipoids; and under specific antigens and reactions, as Lymphogranuloma Venereum; etc.
Anus, microaerophilic hemolytic streptococcus infection of axillas, inguinal and perianal region, 681
Aorta, microscopic pathologic appearance of aorta in treated and untreated cases of syphilitic aortitis, 421
Aortitis, syphilitic, microscopic pathologic appearance of aorta in treated and untreated cases of, 421
Aphthosis: See Stomatitis, aphthous
Apparatus for application of irritant vapors to skin, *173
Appel, B.: Plaint, plea and proposal, 200

Arday, E. J.: Lichen planus; plastic induration of penis (Peyronie's disease), 121
Multiple cutaneous leiomyoma, 114
Neurogenic arthropathy of left knee and ankle; gumma of left ankle; juxta-articular nodules due to syphilis; atrophic arthritis, 116
Armies: See Military Medicine; Recruits: War
Arms: See also Extremities; etc.
basal cell epithelioma of left axilla and left arm, 677
sesame oil tumors, 97
Arnold, H. L., Jr.: Colloid pseudomilium; review of its nomenclature and report of case, *262
Arsenic and Arsenic Compounds: See also Arsphenamines
arsenical dermatitis with superficial epitheliomatosis, 324
arsenical keratoses; arsenical epitheliomas, 681
arsenical keratoses of palms and soles, 138
arsenical pigmentation and hyperkeratosis, 547
arsenical pigmentation and keratoses, 458
blood level and excretion of arsenic following single injections of clorarsen, *297
cerebral reactions associated with massive mapharsen treatment of early syphilis, 204
complications of antisyphilitic therapy in pregnancy, *83
pagetoid epithelioma (scrotum); arsenical keratoses (palms and soles), 319
Therapy: See under Syphilis
Arsphenamines, respiratory allergy from arsphenamines; personal case report, 98
Arteries: See also Aorta; Blood pressure: Embolism; Periarteritis; etc.
Coronary: See Coronary Vessels
Arthritis, arthropathic pustular psoriasis, 224
cutaneous and subcutaneous nodules with juxta-articular infiltrations and rheumatoid symptoms, 425
neurogenic arthropathy of left knee and ankle; gumma of left ankle; juxta-articular nodules due to syphilis; atrophic arthritis, 116
tertiary syphilis; arthropathy of hands and wrists with multiple areas of cystic degeneration of bone, 117
Arthropathy: See Arthritis
Asthma: See also Anaphylaxis and Allergy
allergenic properties of vegetable gums; case of asthma due to tragacanth, 423
respiratory allergy from arsphenamines; personal case report, 98
Athlete's Foot: See Ringworm
Atopy: See Anaphylaxis and Allergy
Atrophoderma: See Atrophy
Atrophy: See also Acrodermatitis chronica atrophicans; and under names of organs and regions
of skin or poikiloderma, 463
of tissues and pigmentation, 666
porokeratosis (Mibelli) associated with cutaneous horn, dystrophy of nails and atrophy of interosseous muscles; report of case, *50
with lichen planus, 334
Avitaminoses: See under Vitamins
Axilla: See also Sweat Glands
basal cell epithelioma of left axilla and left arm, 677
microaerophilic hemolytic streptococcus infection of axillas, inguinal and perianal region, 681
Ayres, S., Jr.: Arsenical keratoses of palms and soles, 138
Cheilitis seborrheica and seborrheic dermatitis of scalp, 136
Contact dermatitis of eyelids of undetermined cause, 326
Fibroma; case for diagnosis, 471
Lichen planus atrophicus, 472
Lupus erythematosus, 472
Multiple extensive ulcerations of legs, 327
Prickle cell epithelioma of lip in young woman, 139

B C G: See under Tuberculosis
Bacteria: See Meningococci; Staphylococci; Streptococci; Tubercle Bacilli; Viruses; etc.
Dysentery: See Dysentery
Leprosy: See Leprosy
Baldness: See Alopecia
Bancroft, I.: Lupus erythematosus; case for diagnosis, 323
Bancroft, J. R.: Keloid, 470
Banyai, A. L.: Changes in finger nails in pulmonary tuberculosis, *306
Barton, R. L.: Fixed drug eruption produced by diphenylhydantoin sodium, *413
Bazin's Disease: See Erythema induratum
Beard: See Hair
Becker, F. T.: Congenital auricular fistula, *520
Becker, S. W.: Dermatitis factitia, neurotic excoriations or tuberculids, 218
Dyskeratosis bullosa (so-called benign familial pemphigus), 217
Melanosis of unusual type, 456
Monilethrix or trichorrhexis nodosa, 331
Sarcoid (miliary), 223
Unusual form of naevus pigmentosus, 228
Bell's Palsy: See Paralysis, facial
Benzyl Benzoate: See Scabies
Bernhardt, E.: Simplified method for staining fungi in skin and in nails, *533
Besnier-Boeck's Disease: See Sarcoidosis
Binjai Tree: See Mango
Biographies:
Alexander Samoilovich Rosenblium; his contribution to fever therapy, *52
Biotin: See Vitamins, B
Bismuth and Bismuth Compounds, intravenous administration of sobisminol solution; toxicity in normal and in syphilitic rabbits, *35
Therapy: See under names of various diseases, i. e., Lupus erythematosus; Syphilis; etc.
Blastomyces, similarity of fungi causing South American blastomycosis (paracoccidioidal granuloma) and North American blastomycosis (Gilchrist's disease), 544
Blastomycosis, 236. See also Coccidioidomycosis; Moniliasis
cutaneous, complicated by meningitis, *88
experimental cutaneous reactions of American blastomycosis in guinea pig, 207
similarity of fungi causing South American blastomycosis (paracoccidioidal granuloma) and North American blastomycosis (Gilchrist's disease), 544
Blepharitis: See Eyelids
Blister Fluid, electrolyte content in pemphigus, 543
production of bullae in skin of duck; experiments with bullae produced by vesicants, *616
production of bullae in skin of duck; preliminary report, *161
Blood, cholesterol, 422
cholesteral values in cases of generalized pruritus, 663
Diseases: See Anemia; Leukemia; etc.
level and excretion of arsenic following single injections of clorarsen, *297
pressure, high; diabetes; hypertensive cardiovascular disease; psoriasis; scleredema, 554
treatment of leg ulcers with blood and concentrated plasma, 420
Bloom, D.: Fixed drug eruption; case for diagnosis, 442
Hypertrophic gingivitis; case for diagnosis, 673
Peculiar marginal blepharitis (due to yellow oxide of mercury), 433
Peculiar pigmentation following injection of tyrosinase in vitiligo, 110
Pemphigus-vulgaris-like eruption following use of sulfonamide compounds, 562
Persistent blepharitis (due to yellow mercuric oxide), 108
Bluefarb, S. M.: Amyloidosis of skin, 337
Colloid milium; case for diagnosis, 454
Localized dermatitis herpetiformis or neurotic excoriations, 237
Lupus erythematosus of eyelids, 233
Lupus vulgaris with pulmonary tuberculosis, 219
Mycosis fungoides, 215
Necrobiosis lipoidica diabeticorum, 226
Vascular disease; case for diagnosis, 353
Boeck Sarcoid: See Sarcoidosis
Bograd, N.: Treatment of tinea with ethyl chloride, *511
Boil, Jericho: See Leishmaniasis

Bones: See also under names of bones
marrow; pulmonary fat embolism following infusions via bone marrow, 205
tertiary syphilis; arthropathy of hands and wrists with multiple areas of cystic degeneration of bone, 117
syphilis with gummas of leg, 242

Book Reviews:
Dictionary of Bio-Chemistry and Related Subjects; W. M. Malisoff, 478
Diseases of Skin; O. S. Ormsby and H. Montgomery, 250
Essentials of Industrial Health; C. O. Sappington, 358
Essentials of Syphilology; R. H. Kampmeier, 357
Formulary and Handbook, The Johns Hopkins Hospital; edited by J. C. Krantz Jr., 686
Fundamentals of Immunology; W. C. Boyd, 477
Handbook of Allergy for Students and Practitioners; W. B. Blanton, 357
Kozhniy leishmanioz (Cutaneous Leishmaniasis); P. V. Kozhevnikov, 476
Methods of Diagnostic Bacteriology; I. G. Schaub and M. K. Foley, 478
1942 Year Book of Dermatology and Syphilology; F. Wise and M. B. Sulzberger, 358
Pênfigo foliáceo (fogo selvagem): Aspectos clinicos e epidemiologicos; J. A. Campos, 141
Primer of Allergy: Guidebook for Those Who Must Find Their Way Through Mazes of This Strange and Tantalizing State; W. T. Vaughan, 141
Synopsis of Diseases of Skin; R. L. Sutton Sr. and R. L. Sutton Jr., 357
War Medicine: Symposium; edited by W. S. Pugh, 142

Bordet-Wassermann Reaction: See Syphilis
Bowen's Disease: See Cancer, precancer
Brain, Diseases: See Encephalitis; etc.
Bromide and Bromine, dermatitis medicamentosa (bromides?), 243
Brooke-Morrow's Disease: See Keratosis follicularis
Bubo, Climatic: See Lymphogranuloma Venereum
Bubonulus: See Lymphogranuloma Venereum
Bullosis: See Vesication
Buphthalmos: See Hydrophthalmos
Burks, J. W.: Histopathologic study of psoriasis, *479
Buschke's Disease: See Scleroderma

Cable Rash: See Chloronaphthalene
Cadden, A. V.: Changes in finger nails in pulmonary tuberculosis, *306
Calamine and neocalamine, 1942, *369
Callosities: See Keratosis
Cancer: See also Epithelioma; Sarcoma; Tumors; and under names of organs and regions, as Lips; etc.
"field-fire" and invasive basal cell carcinoma, basal-squamous type, 661
precancer; Bowen's disease, 213
Cannon, A. B.: Diabetes; hypertensive cardiovascular disease; psoriasis; scleredema, 554
Lichen planus, dermatitis medicamentosa (gold) or peripheral neuritis, 547
Lichen planus, moniliasis or tinea versicolor, 561
Lichenoid exudative discoid dermatitis, 668
Sarcoidosis, 552
Scleroderma, progressive type, 669
Syphilitic gumma of pharynx, 247
Capillaries: See Skin, blood supply
Carbon Dioxide, Solid: See Adenoma
Carbuncle, Malignant: See Anthrax
Carcinoma: See Cancer
Cardiovascular Diseases: See also Heart; etc.
hypertensive; diabetes; psoriasis; scleredema, 554
Cardiovascular System: See Heart; etc.
Carleton, A.: Keratosis follicularis; study of 4 cases, *143
Caro, M. R.: Lupus erythematosus, 222, 331
Lupus erythematosus in young child, 330
Tuberculosis of skin, 218
Carotene: See also Vitamins, A
pityriasis rubra pilaris of familial type; experiences in therapy with carotene and vitamin A, *288
Cat Itch: See Acariasis
Cations: See Ions
Cells: See also Phagocytes and Phagocytosis; Tissue; etc.
mosaic fungus; cholesterol intercellular artefact, *282
virus diseases of skin with reference to elementary and inclusion bodies; variola-vaccinia and molluscum contagiosum, *635
Chancre, multiple chancriform lesions on penis, 474
Charcot's Joints: See Tabes Dorsalis
Chargin, L.: Multiple idiopathic hemorrhagic sarcoma (Kaposi), 244
Vitamin A studies in cases of ichthyosis, *32
Vitamin A studies in cases of keratosis follicularis (Darier's disease), *17
Chaulmoogra Oil, Therapy: See Leprosy
Cheese Itch: See Acariasis
Cheilitis: See under Lips
Chemotherapy: See under Dysentery; Gonorrhea; Impetigo; Skin, diseases; etc.
Chest: See Thorax
Chilblains, normal and injurious effects of cold on skin and underlying tissues; chilblains and allied conditions, 315
Children, false positive serologic tests for syphilis, 208
lymphogranuloma venereum in, 546
prevention of ringworm of scalp in, *533
Chloronaphthalene, halowax acne ("cable rash"); cutaneous eruption in marine electricians due to certain chlorinated naphthalenes and diphenyls, *251
Cholesterol: See also Lipoids
in Blood: See under Blood
mosaic fungus; cholesterol intercellular artefact, *282
Choline and Choline Derivatives, sweat response to acetylcholine, 661
Chorioallantoic Membrane: See Fungi
Chromatophores: See under Melanin
Chu, F. T.: Variations in cutaneous manifestations of vitamin A deficiency from infancy to puberty, *1
Cicatrix, chronic scarring pseudofolliculitis of Negro beard, 312, 539
herpes zoster with scarring and alopecia in 8 year old boy, 667
Cipollaro, A. C.: Basal cell epithelioma, 566
Black hairy tongue, 432
Cutaneous horn with prickle cell epithelioma, 567
Infiltrating basal cell epithelioma, 567
Mycosis fungoides, 428
Seborrheic keratosis with epithelioma, 567
Civatte's Disease: See Poikiloderma
Climacteric: See also Menopause
keratoderma climactericum (Haxthausen); hypoestrogenic keratodermatitis, *270
Clorarsen: See Arsenic and Arsenic Compounds
Clothing, contact dermatitis due to resin-finished facing in cotton underwear, 95
contact dermatitis due to sweat shirt, 239
dermatitis exfoliativa following dermatitis venenata due to shorts, 676, 680
Coal Tar, plaint, plea and proposal, 200
Coccidioides, isolation from soil and rodents, 313
Coccidioidomycosis in wild rodents; method of determining extent of endemic areas, 313
Cohen, D.: Atrophy of skin or poikiloderma, 463
Case for diagnosis, 231
Lichen planus; case for diagnosis, 451
Lupus erythematosus, erythema perstans or erythema elevatum diutinum, 337
Cold: See also Temperature
erythema nodosum, 440
normal and injurious effects of cold on skin and underlying tissues; chilblains and allied conditions, 315
normal and injurious effects of cold on skin and underlying tissues; frostbite, 316
normal and injurious effects on skin and underlying tissues; reactions to cold and injury of normal skin, 315
Cole, A. G.: Cutaneous tests with hen's egg white fractions in atopic infantile eczema, *258

Cole, H. N., Jr.: Chick embryo antigen (lygranum) test for lymphogranuloma venereum; clinical investigation, *379
Poikiloderma atrophicans vasculare (Jacobi), 575
Colitis, multiple ulcers of skin; chronic ulcerative colitis, 551
Colloid pseudomilium; review of its nomenclature and report of case, *262
Combes, F. C.: Prickle cell epithelioma, 248
Tuberculosis or sporotrichosis, 129
Comedones, treatment of acne vulgaris with comedos by monoterminal electrodesiccation, *302
Communicable Diseases: See Meningitis; Syphilis; etc.
Complement Fixation: See under Lymphogranuloma Venereum; Syphilis
Congo Red: See Amyloidosis
Congress: See Societies
Conjunctivitis, fixed eruption and fever from sulfathiazole, *523
Convulsions: See Epilepsy
Coomber, R. B.: Basal cell epithelioma with pigment, secondary to pigmented nevus, 226
Cord, R.: Chick embryo antigen (lygranum) test for lymphogranuloma venereum; clinical investigation, *379
Cornbleet, T.: Arrested acute disseminated lupus erythematosus, 344
Atrophy of skin or poikiloderma, 463
Case for diagnosis, 231
Glomus tumor, 466
Lichen planus; case for diagnosis, 451
Mosaic fungus; cholesterol intercellular artefact, *282
Parapsoriasis; case for diagnosis, 444
Pigmented purpuric lichenoid dermatitis, 467
Trichotillomania, 227
Cornu Cutaneum: See Horns, cutaneous
Coronary Vessels, tobacco skin-reactions in peripheral vascular diseases and coronary artery disease, 206
Correction in transcript of article by Frank H. Moult entitled "Histopathology of Rat Skin in Avitaminosis A" (Arch. Dermat. & Syph. 47: 768 [June] 1943), 540
Cosmetics, acute dermatitis due to hair lacquer, 540
allergic dermatitis caused by hair lacquer products, *656
dermatitis due to nail polish foundation, *197, 419
dermatitis due to nail polish; report of 2 cases, with experimental studies, *612
use of urea in hand creams, *47
Costa, O. G.: Case of keloids of unusual size, *411
Microsporon infection of palpebral and ciliary regions, *65
Severe tropical ulcer, *414
Traumatic marginal alopecia due to traction on hair; comparative study of alopecia liminaris frontalis of Sabouraud, *527
Costello, M. J.: Dermatitis exfoliativa following dermatitis venenata due to shorts, 680
Dermatitis venenata (shorts dermatitis), 676
Dermatomyositis improved by oral administration of dihydrotachysterol, 429
Epidermolysis bullosa and ichthyosis occurring simultaneously, 434
Lymphangioma circumscriptum, 441
Microaerophilic hemolytic streptococcus infection of axillas, inguinal and perianal region, 681
Polycythaemia vera accompanied by acne urticata polycythemica and loss of directional power in fingers of left hand, 430
Tertiary cutaneous syphilis complicated by secondary infection, possibly by microaerophilic hemolytic streptococci, 132
Couperus, M.: Multiple idiopathic hemorrhagic sarcoma, 135
Creams: See Cosmetics
Cysts: See under names of organs and regions, as Synovial Membrane; etc.

Dandruff: See Seborrhea
Darier's Disease: See Keratosis follicularis
Darling's Disease: See Histoplasmosis
Davidson, D. M.: Morphoea guttata, 105
Davis, M. I. J.: Porokeratosis (Mibelli) associated with cutaneous horn, dystrophy of nails and atrophy of interosseous muscles; report of case, *50
Deficiency Diseases: See Vitamins; etc.
Degeneration, Amyloid: See Amyloidosis
colloid, of skin (unilateral), 241
post-traumatic, and hyperplasia, 135
Dentition: See Teeth
Depigmentation: See Pigmentation
Depression-hyposexual-alopecia syndrome, 317
Derbes, V. J.: Yellow dermographia, *310
Dermatitis: See also Acrodermatitis; Eczema; Neurodermatitis; etc.
Acneform: See Acne
Actinica: See under Light
and eczema, 209
Arsphenamine: See Arsphenamines
Atopic: See Eczema; Neurodermatitis
Atrophicans: See Atrophy
Blastomycetic: See Blastomycosis
Contact: See Dermatitis venenata
due to Tyroglyphus longior, Gerv., var. Castellanii, Hirst, in cheese dust, 425
Eczematous: See Eczema
exfoliativa, following dermatitis venenata due to shorts, 676, 680
exfoliativa, secondary to psoriasis, 667
exfoliativa, with leukemoid blood picture indistinguishable from lymphatic leukemia, *42
factitia; actinomycosis or neurotic excoriations, 550, 562
factitia; neurotic excoriations, 550
factitia, neurotic excoriations or tuberculids, 218
factitia or erythema multiforme, 679
fixed eruption, conjunctivitis and fever from sulfathiazole, *523
from exposure to tear gas, 425
halowax acne ("cable rash"); cutaneous eruption in marine electricians due to certain chlorinated naphthalenes and diphenyls, *251
herpetiformis, 236, 684
herpetiformis in child, 210
herpetiformis, localized, or neurotic excoriations, 237
herpetiformis or pemphigus vulgaris, 114
in tattooed areas, 557
lichenoid exudative discoid dermatitis, 668
Medicamentosa: See under Drugs; and under names of drugs, as Arsenic and Arsenic Compounds; Arsphenamines; Bismuth and Bismuth Compounds; Bromide and Bromine; Gold and Gold Compounds; Mercury; etc.
Multiformis: See Dermatitis herpetiformis
Occupational: See Industrial Diseases
petroleum dermatitis; report of 2 cases, *193
repens, 211
Rhus: See Rhus, poisoning
Roentgen Ray: See under Roentgen Rays
Seborrheic: See Seborrhea
Solar: See under Light
toxic, due to ergot, 134
Venenata: See also Plants; Resins; Rhus, poisoning; etc.
venenata; acute dermatitis due to hair lacquer, 540
venenata; allergic dermatitis caused by hair lacquer products, *656
venenata caused by smoke, 425
venenata; contact dermatitis, 685
venenata; contact dermatitis due to resin-finished facing in cotton underwear, 95
venenata; contact dermatitis due to sweat shirt, 239
venenata; contact dermatitis of eyelids of undetermined cause, 326
venenata; dermatitis exfoliativa following dermatitis venenata due to shorts, 676, 680
venenata due to mistletoe, *416
venenata due to nail polish foundation, *197, 419
venenata due to nail polish; report of 2 cases, with experimental studies, *612
Dermatologic societies, directory of, 687
Dermatology, dermatologists in war, 417
military, 99
results of questionnaire on patch tests; analysis of answers by 211 dermatologists and 55 industrial physicians, *514
Dermatomycosis: See Mycosis, cutaneous
Dermatomyositis, case of, 209
improved by oral administration of dihydrotachysterol, 429
Dermatophytosis: See Mycosis, cutaneous

Dermatoses: See Dermatitis; Eczema; Skin, diseases; etc.
Dermographia, phenomenon of "black dermographism," 97
yellow, *310
Diabetes Mellitus; acanthosis nigricans of juvenile type, mild diabetes and obesity, 468
diabetes; hypertensive cardiovascular disease; psoriasis; scleredema, 554
necrobiosis lipoidica diabeticorum with xanthoma cells; report of case, *606
pemphigus; diabetes, 555
skin disturbances in; their relation to vitamin deficiencies, 424
Diachylon: See Eczema
Diathesis, Hemorrhagic: See Purpura; etc.
Diethylstilbestrol: See Estrogens
Dihydrotachysterol: See Hydrotachysterol
Diphenyl: See Phenyl Compounds
Diphenylhydantoin Sodium: See Phenytoin
Director, W.: Fixed eruption, conjunctivitis and fever from sulfathiazole, *523
Directory of dermatologic societies, 687
Ditkowsky, S. E.: Cutaneous tests with hen's egg white fractions in atopic infantile eczema, *258
Dopa: See Hydroxyphenylalanine
Dorne, M.: Arsenical pigmentation and keratoses, 458
Case for diagnosis, 339
Lichen planus with atrophy (?), 334
Lupus erythematosus; case for diagnosis, 457
Lupus erythematosus of eyelid, 339
Pemphigus of mouth improving with acetarsone therapy, 457
Recalcitrant eruption of palms, 446
Schamberg's progressive pigmentary dermatosis or hypostatic purpura, 329
Dostrovsky, A.: Phagedenic ulcer (pyoderma gangraenosum); treatment with sulfapyridine powder and moist chamber therapy, *164
Downing, J. G.: Results of questionnaire on patch tests; analysis of answers by 211 dermatologists and 55 industrial physicians, *514
Driver, J. R.: Poikiloderma atrophicans vasculare (Jacobi), 575
Drugs, Eruption: See also under names of drugs, as Arsenic and Arsenic Compounds; Arsphenamines; Bismuth and Bismuth Compounds; Bromide and Bromine; Gold and Gold Compounds; Mercury; etc.
eruption, fixed; case for diagnosis, 442
eruption, fixed, conjunctivitis and fever from sulfathiazole, *523
Ducks, production of bullae in skin of duck; experiments with bullae produced by vesicants, *616
production of bullae in skin of duck; preliminary report, *161
Duhring's Disease: See Dermatitis herpetiformis
Durand-Nicolas-Favre's Disease: See Lymphogranuloma Venereum
Dust, folliculitis from, *599
Dysentery, bacilli; mechanism of action of certain sulfonamide compounds, 542
Dyskeratosis: See Keratosis
Dystrophy: See Nails

Ear, congenital auricular fistula, *520
herpes zoster oticus treated with omnadin, 546
treatment of otomycosis, 660
Ebert, M. H.: Arthropathic pustular psoriasis, 224
Bowen's disease, 213
Chronic undermining and burrowing ulcer (Meleny), 341
Dermatitis herpetiformis in child, 210
Erythema induratum; case for diagnosis, 212, 343
Gangrene; case for diagnosis, 336
Herpes zoster; case for diagnosis, 450
Lichen planus with recurrent attacks associated with exacerbations of hyperthyroidism, 446
Lupus erythematosus disseminatus subacutus, 452
Lupus erythematosus of lips, 356
Lymphoblastoma or psoriasis, 328
Mycosis fungoides in tumor stage, 214
Ebert, M. H.—Continued
Parkes Weber syndrome; hemangiectatic hypertrophy, 449
Schönlein's purpura or rickettsial infection, 464
So-called myoblastoma, 348
Virus diseases of skin with reference to elementary and inclusion bodies; variola-vaccinia and molluscum contagiosum, *635
Ebert, R. H.: Angioid streaks and pseudoxanthoma elasticum, *75
Eczema and dermatitis, 209
and pruritus in aged, 99
cutaneous tests with hen's egg white fractions in atopic infantile eczema, *258
lead poisoning caused by plaster of lead oleate (diachylon), 661
lichen planus or pityriasis rosea, 439
Seborrheic: See Seborrhea
studies in abnormal human sensitivity to light; prurigo aestivalis, eczema solare and urticaria photogenica; report of 17 cases and review of literature; light sensitivity in prurigo aestivalis, eczema solare and urticaria photogenica; report of experiments, 206
use of urea in hand creams, *47
Edema: See Elephantiasis; and under names of organs

EDITORIALS:
Dermatologists in war, 417

Education, teaching of occupational dermatoses, *153
Elbow, juxta-articular nodes of elbows and palms, 130
Electrocoagulation: See under specific headings
Electrodesiccation: See Acne vulgaris
Electrolytes: See also Ions
electrolyte content of blister fluid in pemphigus, 543
Electropyrexia: See under Scleroderma
Electrosurgery: See under specific headings, as Acne; etc.
Elephantiasis, elephantiasic papulonodular myxedema, *70
of vulva of undetermined cause; furrowed tongue; perlèche, 221
Embolism, pulmonary fat embolism following infusions via bone marrow, 205
Emotions; emotional settings of some attacks of urticaria, 100
Encephalitis, cerebral reactions associated with massive mapharsen treatment of early syphilis, 204
Vegetative: See Acrodynia
Endamoeba Histolytica: See Amebiasis
Engelhardt, H. T.: Yellow dermographia, *310
Enzymes, leukoderma treated with intracutaneous injections of tyrosinase enzyme, 430
vitiligo treated with tyrosinase, 438
Epidermolysis bullosa and ichthyosis occurring simultaneously, 434
bullosa hereditaria, 685
bullosa; report of case, *159
Epidermomycosis; Epidermophytosis: See Moniliasis; Mycosis, cutaneous; Ringworm
Epilepsy, cutaneous nevus with buphthalmos and epilepsy, 664
fixed drug eruption produced by diphenylhydantoin sodium, *413
Epithelioma, arsenical; arsenical keratoses, 681
arsenical dermatitis with superficial epitheliomatosis, 324
basal cell, 437, 566
basal cell, of left axilla and left arm, 677
basal cell, with pigment, secondary to pigmented nevus, 226
case for diagnosis, 470
gumma of mouth with pseudoepitheliomatous hyperplasia, seborrheic dermatitis or psoriasiform parakeratosis of skin, 574
infiltrating basal cell epithelioma, 567
intraepidermal, 322
l'poma-like basal cell epithelioma, *176
multiple benign cystic epithelioma, 102
multiple benign cystic epithelioma and seborrheic keratosis, 102
pagetoid (scrotum); arsenical keratoses (palms and soles), 319
prickle cell, 248

Epithelioma—Continued
prickle cell, of lip in young woman, 139
prickle cell, with cutaneous horn, 567
results of irradiation treatment of cancer of lip; analysis of 636 cases from 1926-1936, 100
with seborrheic keratosis, 567
Ergot, toxic dermatitis due to, 134
Eruptions: See under Arsenic and Arsenic Compounds; Arsphenamines; Drugs; Skin, diseases; etc.
Erysipeloid, *582
Erythema elevatum diutinum, lupus erythematosus or erythema perstans, 337
hyperhidrosis of feet, 538
induratum, 103, 334
induratum and advanced pulmonary tuberculosis, 103
induratum; case for diagnosis, 212, 343
induratum or erythema nodosum, 445
multiforme, 137
multiforme or dermatitis factitia, 679
multiforme perstans recurrens and psoriasis of palms, 435
nodosum; case for diagnosis, 436
nodosum (cold), 440
nodosum; histopathology of Mantoux reaction and erythema nodosum, 318
nodosum or erythema induratum, 445
palmare (red palms), 107
perstans, lupus erythematosus or erythema elevatum diutinum, 337
toxic, fixed type of, 462
Erythredema: See Acrodynia
Erythroderma, Exfoliativum: See Dermatitis exfoliativa
Ichthyosiform: See Ichthyosis
Maculopapular: See Parapsoriasis
Esthiomene: See Lymphogranuloma Venereum
Estradiol: See Estrogens
Estrin, M. M.: Kaposi's sarcoma, 665
Estrogens, effect of estradiol and diethylstilbestrol on atrophic human buccal mucosa, with preliminary report on use of estrogen in management of senile gingivitis, 424
effect of estradiol locally applied to abnormal skin, *188
keratoderma climactericum (Haxthausen): hypoestrogenic keratodermatitis, *270
sesame oil tumors, 97
treatment of essential pruritus and kraurosis vulvae, 424
Ethylamine: See Amines
Ethyl Chloride: See Tinea
Extremities: See also Arms; etc.
Blood Supply: See also Raynaud's Disease; Thrombophlebitis; etc.
blood supply; tobacco skin-reactions in peripheral vascular diseases and coronary artery disease, 206
blood supply; vascular disease; case for diagnosis, 353
normal and injurious effects of cold on skin and underlying tissues; chilblains and allied conditions, 315
paralysis; paresis following herpes zoster, 314
Parkes Weber syndrome; hemangiectatic hypertrophy, 449
Eyelashes, microsporon infection of palpebral and ciliary regions, *65
seborrheic dermatitis of eyelids and canities of eyelashes, 324
Eyelids, contact dermatitis of undetermined cause, 326
gumma of upper lip, lower right eyelid and hard palate, 118
lupus erythematosus of, 233, 339
microsporon infection of palpebral and ciliary regions, *65
peculiar marginal blepharitis (due to yellow oxide of mercury), 433
persistent blepharitis (due to yellow mercuric oxide), 108
persistent marginal blepharitis (due to yellow mercuric oxide), 443
seborrheic dermatitis of eyelids and canities of eyelashes, 324
Eyes: See also Vision; and under names of special structures and diseases of eye, as Retina; etc.
syphilis, treatment of, 100
syphilitic gumma of, 672
Face, diseases; clinical use of penicillin, 100
Paralysis: See Paralysis, facial
tuberculid, rosacea-like, 555
Fat: See also Lipoids
adiponecrosis neonatorum, 113
lecithin feeding in syndrome of psoriasis; chemical studies of relationship of lecithin feeding to fat metabolism, 544
metabolism in acne vulgaris, 207
Favosa: See Favus
Favre-Nicolas' Disease: See Lymphogranuloma Venereum
Favus of glabrous skin and mucous membranes or moniliasis of skin and mucous membranes, 119
Feer's Disease: See Acrodynia
Feet: See Foot
Feller, H. B.: Lupus erythematosus; case for diagnosis, 109
Felsher, I. M.: Lupus erythematosus in young child, 330
Tuberculosis of skin, 218
Fever, Alexander Samoilovich Rosenblium; his contribution to fever therapy, *52
fixed eruption, conjunctivitis and fever from sulfathiazole, *523
Rocky Mountain Spotted: See Rocky Mountain Spotted Fever
Therapeutic: See Scleroderma; Syphilis; etc.
Fibroma, case for diagnosis, 471
Fifth Venereal Disease: See Granuloma inguinale
Fingers and Toes: See also Foot; Hand
Nails: See Nails
polycythaemia vera accompanied by acne urticata polycythemica and loss of directional power in fingers of left hand, 430
Finkle, T. H.: Adenoma sebaceum treated by solid carbon dioxide, *198
Finnerud, C. W.: Basal cell epithelioma with pigment, secondary to pigmented nevus, 226
Case for diagnosis subsequently proved to be 1 of syphilis, 453, 469
Fistula, congenital auricular, *520
Folliculitis, actual causes of certain occupational dermatoses; study of 527 cases with reference to dermatoses due to mineral oils, *591, *596
case for diagnosis, 565
decalvans; lichen planus et atrophicus (folliculitis decalvans and lichen spinulosus, Little and McCafferty), 669
ulerythematosa reticulata, 673
Foot: See also Ankle; Fingers and Toes; etc.
hyperhidrosis of feet, 538
von Recklinghausen's disease with involvement of scalp, palms and soles; report of case, *656
Ford, W. K.: Scabies or syphilis, 453
Superficial nonulcerative syphilis; case for diagnosis, 452
Foreign Bodies; foreign body reaction; case for diagnosis, 448
Fourth Venereal Disease: See Lymphogranuloma Venereum
Fox, H.: Actinomycosis or neurotic excoriations, 550, 562
Lupus vulgaris hypertrophicus, 549
Frank, L. J.: Herpes zoster with scarring and alopecia in 8 year old boy, 667
Franks, A. G.: Cutaneous blastomycosis complicated by meningitis, *88
Porokeratosis (Mibelli) associated with cutaneous horn, dystrophy of nails and atrophy of interosseous muscles; report of case, *50
Fraser, J. F.: Exfoliative dermatitis with leukemoid blood picture indistinguishable from lymphatic leukemia, *42
Frazier, C. N.: Variations in cutaneous manifestations of vitamin A deficiency from infancy to puberty, *1
Freeman, C. W.: Vitiligo of upper lip in players of wind instruments, *605
Frei Reaction: See Lymphogranuloma Venereum
Frost, K.: Post-traumatic degeneration and hyperplasia, 135
Seborrheic dermatitis of eyelids and canities of eyelashes, 324
Frostbite: See also Chilblains
normal and injurious effects of cold on skin and underlying tissues; frostbite, 316

Fruchtbaum, L. M.: Dermatitis medicamentosa (bromides?), 243
Fungi: See also Actinomycosis; Blastomycosis; Moniliasis; Mycosis; Ringworm; etc.
inhibitory effect of monochlormercuricarvacrol on growth of pathogenic fungi, *497
mosaic; cholesterol intercellular artefact, *282
simplified method for staining fungi in skin and in nails, *533
virulence of strains of phialophora verrucosa determined by inoculating chorioallantoic membranes of chick embryos, 545
Furunculosis, etiology and treatment, 424

Gahan, E.: Incidence of psoriasis among population at large, *305
Gammel, J. A.: Lichen sclerosis et atrophicus: case for diagnosis, 115
Gangrene, case for diagnosis, 336
Garb, J.: Mycosis fungoides with bullous lesions; report of case resistant to roentgen and arsenical therapy; effects of empiric therapy, partly based on laboratory investigations, *359
Gas, poisoning; dermatitis from exposure to tear gas, 425
poisoning; production of bullae in skin of duck; experiments with bullae produced by vesicants, *616
Gauvain, E. A.: Lupus vulgaris, 242
Roentgen dermatitis, 244
Genitals: See Genitourinary Tract; and under names of genitals, as Penis; etc.
Genitourinary Tract, tuberculosis of cutis and hypoderm associated with genitourinary tuberculosis and chronic pulmonary tuberculosis, 249
Georgi, C. E.: Inhibitory effect of monochlormercuricarvacrol on growth of pathogenic fungi, *497
Gilchrist's Disease: See Blastomycosis
Gingivitis: See Gums
Glick, A. W.: Vitamin A studies in cases of ichthyosis, *32
Vitamin A studies in cases of keratosis follicularis (Darier's disease), *17
Goeckerman, W. H.: Arsenical dermatitis with superficial epitheliomatosis, 324
Gold and Gold Compounds, lichen planus, dermatitis medicamentosa (gold) or peripheral neuritis, 547
Therapy: See under Lupus erythematosus; etc.
Goldman, L.: Acquired hypersensitivity to chlorinated ethylamine vesicants; report of case, *15
Production of bullae in skin of duck; experiments with bullae produced by vesicants, *616
Production of bullae in skin of duck; preliminary report, *161
Gonorrhea: See also Venereal Diseases
chemotherapeutic prophylaxis with sulfonamide drugs, 420
migrating thrombophlebitis following gonorrhea and medication with sulfonamide drugs, 128
Good, C. K.: Halowax acne ("cable rash"); cutaneous eruption in marine electricians due to certain chlorinated naphthalenes and diphenyls, *251
Goodfriend, J.: Livedo reticularis (syphilis), 216
Goodman, H.: Calamine and neocalamine, 1942, *369
Gordon Test: See Hodgkin's Disease
Grace, A. W.: Beneficial effect of smallpox vaccine on recurrent aphthous ulcers of mouth and tongue, *151
Chick embryo antigen (lygranum) test for lymphogranuloma venereum, 659
Complement fixation test for lymphogranuloma venereum; results obtained with its use, *619
Granuloma annulare, 461
annulare, contribution to study of, 101
cholesterinicum subcutaneum; new form of localized lipoidosis, 544
Fungoides: See Mycosis fungoides
Inguinale: See also Lymphogranuloma Venereum
inguinale, bubonulus in, *494
inguinale; late latent syphilis, 112
inguinale, lymphogranuloma venereum or tuberculosis, 351
Granuloma—Continued
Malignant: See Hodgkin's Disease
Paracoccidioidal: See Blastomycosis
Venereal: See Granuloma Inguinale; Lymphogranuloma Venereum
Greenbaum, S. S.: Chronic scarring pseudofolliculitis of Negro beard, 312
Groin, microaerophilic hemolytic streptococcus infection of axillas, inguinal and perianal region, 681
Gum: See Tragacanth
Gumma: See Ankle; Eyelids; Leg; Pharynx; etc.
Gums, effect of estradiol and diethylstilbestrol on atrophic human buccal mucosa, with preliminary report on use of estrogen in management of senile gingivitis, 424
hypertrophic gingivitis; case for diagnosis, 673

Habermann's Disease: See Parapsoriasis
Hair: See also Alopecia
abnormalities; monilethrix or trichorrhexis nodosa, 331
acute dermatitis due to hair lacquer, 540
allergic dermatitis caused by hair lacquer products, *656
Diseases: See also Alopecia
diseases; piedra, 473
diseases; trichorrhexis nodosa or monilethrix, 331
effect of androgen on sebaceous glands of human skin, *601
ingrown; chronic scarring pseudofolliculitis of Negro beard, 312, 539
Plucking: See Trichotillomania
traumatic marginal alopecia due to traction on hair; comparative study of alopecia liminaris frontalis of Sabouraud, *527
Hall, A. F.: Intraepidermal epithelioma, 322
Hallopeau's Disease: See Acrodermatitis
Halloran, C. R.: Case for diagnosis, 470
Lupus vulgaris, 137
Lymphoblastoma; case for diagnosis, 140
Halowax: See Chloronaphthalene
Hand, E. A.: Scabies or syphilis, 453
Superficial nonulcerative syphilis; case for diagnosis, 452
Hand: See also Fingers and Toes
juxta-articular nodes of elbows and palms, 130
neurofibromatosis of palms and wrists; keloids, 246
polycythaemia vera accompanied by acne urticata polycythemica and loss of directional power in fingers of left hand, 430
recalcitrant eruption of palms, 446
von Recklinghausen's disease with involvement of scalp, palms and soles; report of case, *656
Hand Creams: See Cosmetics
Hansen's Disease: See Leprosy
Hanzlik, P. J.: Intravenous administration of sobisminol solution; toxicity in normal and in syphilitic rabbits, *35
Haxthausen's Disease: See Keratosis
Hazen, H. H.: Vitiligo of upper lip in players of wind instruments, *605
Head, angiomas of, 561
Heart, effect of Schaumann's disease on heart and its mechanism, 546
Hecht, R.: Cutaneous tests with hen's egg white fractions in atopic infantile eczema, *258
Hedge, H. M.: Pemphigus erythematosus, 330
Heisel, E. B.: Adiponecrosis neonatorum, 113
Chick embryo antigen (lygranum) test for lymphogranuloma venereum; clinical investigation, *379
Granuloma inguinale; late latent syphilis, 112
Gumma of upper lip, lower right eyelid and hard palate, 118
Pemphigus vulgaris or dermatitis herpetiformis, 114
Tertiary syphilis; athropathy of hands and wrists with multiple areas of cystic degeneration of bone, 117
Hemangiectasis, Parkes Weber syndrome; hemangiectatic hypertrophy, 449
Hemangioma, cavernous, of scalp treated by interstitial irradiation with low intensity radium needles, 573
Hemopoietic System, Diseases: See Anemia; Leukemia; etc.
Henington, V. M.: Complications of antisyphilitic therapy in pregnancy, *83

Henning, H. B.: Blood level and excretion of arsenic following single injections of clorarsen, *297
Henningsen, A. B.: Acanthosis nigricans of juvenile type, mild diabetes and obesity, 468
Kaposi's sarcoma, 466
Pemphigus vulgaris, 469
Herpes, effect of temperature on growth and survival of myxoma, herpes and vaccinia viruses in tissue culture, 542
zoster, case for diagnosis, 227, 450
zoster oticus treated with omnadin, 546
zoster, paresis following, 314
zoster traumaticus, *581
zoster with scarring and alopecia in 8 year old boy, 667
Hetreed, F. W.: Amyloidosis cutis, 224
Erythema induratum (?), 334
Fixed type of toxic erythema, 462
Lupus vulgaris, 340
Urticaria pigmentosa; early syphilis (?), 460
Hidradenitis: See Sweat Glands
Hinton Test: See Syphilis
Histoplasmosis, specific cutaneous reactions with histoplasmosis; preliminary report of another case, 97
Hodgkin's Disease, ulcerative, 104
with cutaneous manifestations, 106
with specific lesions appearing first in skin, 660
Hollander, A.: Allergic dermatitis caused by hair lacquer products, *656
Acanthosis nigricans; is it form of avitaminosis? *650
Hookworm Infection, severe tropical ulcer, *414
Hormones: See Androgens; Estrogens; etc.
Horns, cutaneous; porokeratosis (Mibelli) associated with cutaneous horn, dystrophy of nails and atrophy of interosseous muscles; report of case, *50
cutaneous, with prickle cell epithelioma, 567
Houston, M.: Vitiligo, *400
Howell, J. B.: Evaluation of measures for prevention of ivy dermatitis, *373
Hu, C. K.: Variations in cutaneous manifestations of vitamin A deficiency from infancy to puberty, *1
Hubler, W. R.: Amebiasis cutis, 571
Chick embryo antigen (lygranum) test for lymphogranuloma venereum; clinical investigation, *379
Favus of glabrous skin and mucous membranes or moniliasis of skin and mucous membranes, 119
Gumma of mouth with pseudoepithelioma tous hyperplasia, seborrheic dermatitis or psoriasiform parakeratosis of skin, 574
Localized scleroderma, 568
Rhinophyma, 570
Hydroa aestivale; photosensitization dermatitis (hydroa aestivale or allergic dermatitis), 325
Hydrocystoma, experimentally induced disappearance and reappearance of lesions of, 205
Hydrophthalmos, cutaneous nevus with buphthalmos and epilepsy, 664
Hydrotachysterol, dermatomyositis improved by oral administration of dihydrotachysterol, 429
Hydroxyphenylalanine, cutaneous melanoblasts as studied with paraffin-dopa technic, 662
Hypercholesteremia: See Blood, cholesterol
Hyperemia: See Skin, blood supply
Hyperkeratosis: See Keratosis
Hyperpigmentation: See Pigmentation
Hyperpyrexia: See under Scleroderma; Syphilis; etc.
Hypertension: See Blood pressure, high
Hyperthyroidism: See under Thyroid
Hypertrophy, hemangiectatic; Parkes Weber syndrome, 449
Hypophysis: See Pituitary Body
Hypotrichosis: See Alopecia

Ichthyosis and epidermolysis bullosa occurring simultaneously, 434
congenital ichthyosiform erythroderma, 458
vitamin A studies in cases of, *32
Icterus: See Jaundice
Immunity: See Anaphylaxis and Allergy; etc.
Impetigo contagiosa, bacteriology of, 101
contagiosa, treatment with new physical form of sulfathiazole, 97
Impotence, depression-hyposexual-alopecia syndrome, 317
Indians, American; sporotrichosis following mosquito bite; description of lesions in girl of Indian and French descent, *525
Industrial Diseases, actual causes of certain occupational dermatoses; study of 527 cases with reference to dermatoses due to mineral oils, *579
course of lectures on occupational dermatoses, 536
dermatitis in tattooed areas, 557
halowax acne ("cable rash"); cutaneous eruption in marine electricians due to certain chlorinated naphthalenes and diphenyls, *251
petroleum dermatitis; report of 2 cases, *193
second annual meeting of Consulting Staff of Dermatoses Investigations Section, 202
teaching of occupational dermatoses, *153
vitiligo of upper lip in players of wind instruments, *605
Industry and Occupations, results of questionnaire on patch tests; analysis of answers by 211 dermatologists and 55 industrial physicians, *514
Infectious Diseases: See Syphilis; etc.
Inguinal Region: See Groin
Insects: See Mosquitoes
Instruments: See Apparatus
Insulin: See also Diabetes Mellitus
hypersensitivity with desensitization; report of case, 423
Intelligence and pediculosis capitis in W. A. A. F. recruits, 318
Ions, removal of divalent cations from solution by beef heart antigens, 206
Irgang, S.: Lupus erythematosus tumidus superficialis; treatment with tuberculin; report of case, *60
Itching: See Pruritus
Ivy Poisoning: See Rhus, poisoning

Jacobi-Petges' Disease: See Poikiloderma atrophicans vasculare
Jacobson, H. P.: Photosensitization dermatitis (hydroa aestivale or allergic dermatitis), 325
Jaenicke, K.: Vitiligo in 3 siblings, *519
Jaundice, yellow dermographia, *310
Jensen, A. K.: Case for diagnosis, 475
Cheilitis solaris, 319
Erythema multiforme, 137
Jericho Boil: See Leishmaniasis
Johnson-Stevens Disease: See Erythema multiforme
Junqueira, M. A.: Traumatic marginal alopecia due to traction on hair; comparative study of alopecia liminaris frontalis of Sabouraud, *527
Juxta-Articular Nodes: See Nodes

Kahn Test: See also under Syphilis
sensitivity and specificity of Kahn reaction in temperature range from 37° C. through 56° C. with syphilitic sera with consideration of value of Kahn verification test in syphilis, 204
Kala-Azar: See Leishmaniasis
Kalz, F.: Syphilitic juxta-articular nodules, *626
Kampmeier, R. H.: Blood level and excretion of arsenic following single injections of clorarsen, *297
Kaposi's Sarcoma: See Sarcoma
Keil, H.: Contact dermatitis due to resin-finished facing in cotton underwear, 95
Dermatitis due to nail polish; report of 2 cases, with experimental studies, *612
Keloid, 470. See also Cicatrix
case of keloids of unusual size, *411
extensive postacne keloids, 431
neurofibromatosis of palms and wrists, 246
Kennedy, C. B.: Complications of antisyphilitic therapy in pregnancy, *83
Keratoderma: See Keratosis
Keratosis: See also Ichthyosis
arsenical; arsenical epitheliomas, 681
arsenical, of palms and soles, 138
arsenical pigmentation and hyperkeratosis, 547
arsenical pigmentation and keratoses, 458
dyskeratosis bullosa (so-called benign familial pemphigus), 217
dyskeratosis congenita with pigmentation, dystrophia unguis and leukokeratosis oris (Cole and others), 560

Keratosis—Continued
follicularis; study of 4 cases, *143
follicularis; vitamin A studies in cases of keratosis follicularis (Darier's disease), *17
gumma of mouth with pseudoepitheliomatous hyperplasia, seborrheic dermatitis or psoriasiform parakeratosis of skin, 574
keratoderma climactericum, 683
keratoderma climactericum (Haxthausen); hypoestrogenic keratodermatitis, *270
pagetoid epithelioma (scrotum); arsenical keratoses (palms and soles), 319
porokeratosis (Mibelli) associated with cutaneous horn, dystrophy of nails and atrophy of interosseous muscles; report of case, *50
seborrheic, and multiple benign cystic epithelioma, 102
seborrheic, with epithelioma, 567
variations in cutaneous manifestations of vitamin A deficiency from infancy to puberty, *1
Kesler, R. L.: Erythema nodosum or erythema induratum, 445
Kesten, B. M.: Neurofibromatosis of palms and wrists; keloids, 246
Kingsbury, J.: Melanoma, 555
Klauder, J. V.: Actual causes of certain occupational dermatoses; study of 527 cases with reference to dermatoses due to mineral oils, *579
Teaching of occupational dermatoses, *153
Kline Test: See under Syphilis
Knee, neurogenic arthropathy of left knee and ankle; gumma of left ankle; juxta-articular nodules due to syphilis; atrophic arthritis, 116
post-traumatic degeneration and hyperplasia, 135
Knoll, M.: Multiple idiopathic hemorrhagic sarcoma, 135
Kraurosis: See Vulva
Krause, M. E.: Adiponecrosis neonatorum, 113
Chick embryo antigen (lygranum) test for lymphogranuloma venereum; clinical investigation, *379
Granuloma inguinale; late latent syphilis, 112
Gumma of upper lip, lower right eyelid and hard palate, 118
Pemphigus vulgaris or dermatitis herpetiformis, 114
Tertiary syphilis; arthropathy of hands and wrists with multiple areas of cystic degeneration of bone, 117

Labia Majora: See Vulva
Lacquer, acute dermatitis due to hair lacquer, 540
allergic dermatitis caused by hair lacquer products, *656
Nail Lacquer: See Cosmetics
Larynx, diseases; specific cutaneous reactions with histoplasmosis; preliminary report of another case, 97
Laymon, C. W.: Arsenical keratoses; arsenical epitheliomas, 681
Lichen sclerosus et atrophicus, 681
Lead poisoning caused by plaster of lead oleate (diachylon), 661
Leavitt, H.: Lupus vulgaris, 242
Lecithin: See Psoriasis
Lectures, course on occupational dermatoses, 536
Legs: See also Extremities; Foot
gummas of leg with syphilis of bones, 242
Ulcers: See Ulcers
Leiomyoma, 248
multiple cutaneous leiomyoma, 114
Leishmaniasis, oral pharyngeal leishmaniasis treated with pentamidine; 2 cases, 314
vaccination against Jericho boil, 314
Leprosy, chaulmoogra oil in treatment, 313
correlation of its clinical, pathologic, immunologic and bacteriologic aspects, 423
lepra; case for diagnosis, 557
Leukemia, exfoliative dermatitis with leukemoid blood picture indistinguishable from lymphatic leukemia, *42
lymphatic, and idiopathic hemorrhagic sarcoma, 566
lymphatic, chronic (cutaneous leukemid), 671
Leukemid: See Leukemia, lymphatic
Leukocytes: See Leukemia; etc.
Leukoderma: See Vitiligo
Leukokeratosis: See Keratosis
Leukopathia: See Vitiligo
Levin, A. A.: Pityriasis rubra pilaris of familial type; experiences in therapy with carotene and vitamin A, *288
Levin, B.: Cutaneous tests with hen's egg white fractions in atopic infantile eczema, *258
Lewis, G. M.: Erythema multiforme or dermatitis factitia, 679
Erythema nodosum; case for diagnosis, 436
Erythema nodosum (cold), 440
Lichen nitidus, 442
Myxomatous degeneration; synovial cysts, 436
Neurotic excoriations, 550
Pustular psoriasis; case for diagnosis, 678
Lewisite: See Gas, poisoning
Libman-Sacks Disease, urinary sediment in visceral angiitis (periarteritis nodosa, lupus erythematosus, Libman-Sacks "disease"), 422
Lice: See Pediculosis
Lichen: lichenoid exudative discoid dermatitis, 668
nitidus, 442
pigmented purpuric lichenoid dermatitis, 467
planus, 103, 104, 451
planus atrophicus, 472
planus, chronic recurring, 349
planus, dermatitis medicamentosa (gold) or peripheral neuritis, 547
planus et atrophicus (folliculitis decalvans and lichen spinulosus, Little and McCafferty), 669
planus linearis, 680
planus, moniliasis or tinea versicolor, 561
planus, pityriasis rosea or eczema, 439
planus; plastic induration of penis (Peyronie's disease), 121
planus, recurrent, 447
planus with atrophy (?), 334
planus with recurrent attacks associated with exacerbations of hyperthyroidism, 446
ruber planus; effect of certain vehicles and of salt solutions on reactive hyperemia in skin; application in case of lichen ruber planus, 545
sclerosus et atrophicus, 115, 430, 433, 681
spinulosus; lichen planus et atrophicus (folliculitis decalvans and lichen spinulosus, Little and McCafferty), 669
striatus; case for diagnosis, 449
Urticatus: See Urticaria papulosa
Lichenification: See Neurodermatitis
Light, cheilitis solaris, 319
photodynamic effects in dermatology, 660
photosensitization dermatitis (hydroa aestivale or allergic dermatitis), 325
solar urticaria, 229
studies in abnormal human sensitivity to light; passive transfer of light hypersensitivity in prurigo aestivalis, 543
studies in abnormal human sensitivity to light; photoallergic concept of prurigo aestivalis, 543
studies in abnormal human sensitivity to light; prurigo aestivalis, eczema solare and urticaria photogenica; report of 17 cases and review of literature; light sensitivity in prurigo aestivalis, eczema solare and urticaria photogenica; report of experiments, 206
sunburn and para-aminobenzoic acid, 661
Lindsay, H. C. L.: Multiple chancriform lesions of penis, 474
Parapsoriasis, 473
Toxic dermatitis due to ergot, 134
Lingua: See Tongue
Linseed oil dermatitis, *597
Lipids: See Lipoids
Lipoidosis: See Lipoids; Xanthoma
Lipoids: See also Cholesterol; Fat
new form of localized lipoidosis; granuloma cholesterinicum subcutaneum, 544
Lipoma-like basal cell epithelioma, *176
Lips, cancer, results of irradiation treatment; analysis of 636 cases from 1926-1936, 100
cheilitis seborrheica and seborrheic dermatitis of scalp, 136
cheilitis solaris, 319
elephantiasis of vulva of undetermined cause; furrowed tongue; perlèche, 221
gumma of upper lip, lower right eyelid and hard palate, 118
lupus erythematosus of, 356

Lips—Continued
prickle cell epithelioma of lip in young woman, 139
psoriasis (resistant); perlèche; scrotal tongue, 460
vitiligo of upper lip in players of wind instruments, *605
Livedo reticularis, 127
reticularis (syphilis), 216
Loewinsohn, E.: Lichen striatus; case for diagnosis, 449
Moniliasis; case for diagnosis, 455
Lowenfish, F. P.: Tuberculosis of cutis and hypoderm associated with genitourinary tuberculosis and chronic pulmonary tuberculosis, 249
Ludueña, F. P.: Intravenous administration of sobisminol solution; toxicity in normal and in syphilitic rabbits, *35
Luetin Test: See under Syphilis
Lungs, Tuberculosis: See Tuberculosis, pulmonary
Luotest: See under Syphilis
Lupoid: See Sarcoidosis
Lupus: See also Tuberculosis
erythematosus, 222, 472
erythematosus, acute disseminated, 122
erythematosus, acute disseminated, arrested, 344
erythematosus, acute disseminated, medical aspects of, 207
erythematosus, acute, nature of, 207
erythematosus and pulmonary tuberculosis, 103
erythematosus; case for diagnosis, 109, 323, 331, 457
erythematosus disseminatus subacutus, 452
erythematosus, erythema perstans or erythema elevatum diutinum, 337
erythematosus in young child, 330
erythematosus of eyelids, 233, 339
erythematosus of face associated with nodular infiltrations of back, 577
erythematosus of lips, 356
erythematosus or circumscribed scleroderma, 569
erythematosus or sycosis vulgaris, 675
erythematosus timidus superficialis; treatment with tuberculin; report of case, *60
erythematosus, urinary sediment in visceral angiitis (periarteritis nodosa, lupus erythematosus, Libman-Sacks "disease"), 422
vulgaris, 137, 242, 340
vulgaris hypertrophicus, 549
vulgaris with pulmonary tuberculosis, 219
Lygranum: See Lymphogranuloma Venereum
Lymph Nodes: See also Leukemia, lymphatic; etc.
dermatopathic lymphadenitis; focal granulomatous lymphadenitis associated with chronic generalized skin disorders, 206
exfoliative dermatitis with leukemoid blood picture indistinguishable from lymphatic leukemia, *42
sporotrichosis; localized lymphatic type, 578
Lymphadenitis: See Lymph Nodes
Lymphangioma circumscriptum, 441
Lymphedema: See Elephantiasis; Penis
Lymphoblastoma, 214
case for diagnosis, 140, 335
or mycosis fungoides, 234
or psoriasis, 328
resembling neurodermatitis, 437
Lymphogranuloma, Hodgkin's: See Hodgkin's Disease
Inguinale: See Granuloma inguinale; Lymphogranuloma Venereum
Schaumann's: See Sarcoidosis
Lymphogranuloma Venereum, bubonulus in granuloma inguinale, *494
chick embryo antigen (lygranum) test, 659
chick embryo antigen (lygranum) test; clinical investigation, *379
complement fixation for lymphogranuloma venereum and for psittacosis with Frei reactions among pneumonia patients, 541
complement fixation test for; results obtained with its use, *619
evaluation of yolk sac antigen for Frei test, 421
further evidence concerning specificity of lymphogranuloma venereum complement-fixation test in syphilis, 421
granuloma inguinale, lymphogranuloma venereum or tuberculosis, 351
Lymphogranuloma Venereum—Continued
in children, 546
visible forms and possible life cycle of virus, 318
Lymphopathia Venereum: See Lymphogranuloma Venereum
Lynch, F. W.: Epidermolysis bullosa hereditaria, 685
Keratoderma climactericum (Haxthausen); hypoestrogenic keratodermatitis, *270

Machacek, G. F.: Chronic lymphatic leukemia (cutaneous leukemid), 671
Orthostatic lymphedema of penis and scrotum, 670
McNary, R. R.: Acquired hypersensitivity to chlorinated ethylamine vesicants; report of case, *15
Apparatus for application of irritant vapors to skin, *173
Madden, J. F.: Erythema induratum, 103
Erythema induratum and advanced pulmonary tuberculosis; case for diagnosis, 103
Lichen planus, 103
Lichen urticatus, 102
Lupus erythematosus and pulmonary tuberculosis, 103
Urticaria pigmentosa, 102
Majocchi's Disease: See Purpura annularis telangiectodes
Mango, dermatitis venenata caused by smoke, 425
Manting, G.: Sporotrichosis following mosquito bite; description of lesions in girl of Indian and French descent, *525
Mantoux Reaction: See Tuberculosis
Mapharsen: See Arsenic and Arsenic Compounds; Syphilis
Medicine, Military: See Military Medicine
Melanin: See also Melanoma; Pigmentation
pigmentation; cutaneous melanoblasts as studied with paraffin-dopa technic, 662
pigmentation; melanosis of unusual type, 456
Melanoblasts: See Melanin, pigmentation
Melanoma, 555
nevus, melanoma or onychomycosis, 134
Melanosis: See Melanin, pigmentation
Meningitis: See also Meningococci
cutaneous blastomycosis complicated by, *88
Meningococci: See also Meningitis
infection, skin manifestations of, 662
Menopause, effect of estradiol and diethylstilbestrol on atrophic human buccal mucosa, with preliminary report on use of estrogen in management of senile gingivitis, 424
keratoderma climactericum, 683
Mental Diseases: See also Neuroses and Psychoneuroses
Alexander Samoilovich Rosenblium; his contribution to fever therapy, *52
Mercury, peculiar marginal blepharitis (due to yellow oxide of mercury), 433
persistent blepharitis (due to yellow mercuric oxide), 108
persistent marginal blepharitis (due to yellow mercuric oxide), 443
Metacresyl Acetate: See Mycosis
Metals: See also Mercury; etc.
phenomenon of "black dermographism," 97
Mibelli's Disease: See Keratosis
Michelson, H. E.: Aleukemic reticuloendotheliosis, 683
Dystrophy of nails, 684
Raynaud's disease and generalized progressive scleroderma, 682
Michelson, W.: Perifolliculitis capitis abscendens et suffodiens, 226
Microsporosis; microsporon infection of palpebral and ciliary regions, *65
Military Medicine: See also Gas, poisoning; Recruits; etc.
control of venereal disease in army, 101
epidermolysis bullosa; report of case, *159
military dermatology, 99
preliminary intensive treatment of primary syphilis by daily injection of arsenicals, 204
skin diseases in war, 317
treatment of scabies in wartime, *370
Milium, Colloid: See Skin, colloid degeneration
Miller, E. M.: Sporotrichosis, 240
Miller, J. L.: Leiomyoma, 248
Syphilitic gumma of eye, 672

Mineral Oil: See Petrolatum, liquid
Mirsky, I. A.: Production of bullae in skin of duck; experiments with bullae produced by vesicants, *616
Production of bullae in skin of duck; preliminary report, *161
Miskjian, H. G.: Lupus erythematosus of face associated with nodular infiltrations of back, 577
Mistletoe, dermatitis venenata due to, *416
Mitchell, J. H.: Amyloidosis cutis, 224
Chronic recurring lichen planus, 349
Erythema induratum (?), 334
Erythema nodosum or erythema induratum, 445
Fixed type of toxic erythema, 462
Lupus vulgaris, 340
Mycosis fungoides, 353
Mycosis fungoides; case for diagnosis, 214
Report of cultures of sporotrichosis, 341
Sporotrichosis, 240
Tetryl dermatitis, 346
Urticaria pigmentosa; early syphilis (?), 460
Molds: See Fungi
Moles: See Nevi
Molluscum contagiosum; virus diseases of skin with reference to elementary and inclusion bodies; variola-vaccinia and molluscum contagiosum, *635
Monilethrix: See Hair, abnormalities
Moniliasis, case for diagnosis, 455
favus of glabrous skin and mucous membranes or moniliasis of skin and mucous membranes, 119
lichen planus, moniliasis or tinea versicolor, 561
Monochlormercuricarvacrol, inhibitory effect of monochlormercuricarvacrol on growth of pathogenic fungi, *497
Montgomery, A. H.: Massive angioma cavernosum treated with solid carbon dioxide, 670
Montgomery, H.: Histopathologic study of psoriasis, *479
Moore, M.: Sporotrichosis following mosquito bite; description of lesions in girl of Indian and French descent, *525
Morphea: See Scleroderma
Mosquitoes, severe tropical ulcer, *414
sporotrichosis following mosquito bite; description of lesions in girl of Indian and French descent, *525
Mouth: See also Gums; Stomatitis; etc.
beneficial effect of smallpox vaccine on recurrent aphthous ulcers of mouth and tongue, *151
dyskeratosis congenita with pigmentation, dystrophia unguis and leukokeratosis oris (Cole and others), 560
effect of estradiol and diethylstilbestrol on atrophic human buccal mucosa, with preliminary report on use of estrogen in management of senile gingivitis, 424
gumma of mouth with pseudoepitheliomatous hyperplasia, seborrheic dermatitis or psoriasiform parakeratosis of skin, 574
oral pharyngeal leishmaniasis treated with pentamidine; 2 cases, 314
pemphigus improving with acetarsone therapy, 457
prosthetic ulcer of, 314
tuberculosis orificialis, 235
Mucous Membrane: See also Lips; Mouth; Nose; etc.
favus of glabrous skin and mucous membranes or moniliasis of skin and mucous membranes, 119
Music, vitiligo of upper lip in players of wind instruments, *605
Mycobacterium Leprae: See under Leprosy
Mycosis: See also Actinomycosis; Blastomycosis; Moniliasis; Nails; Ringworm; Tinea; etc.
cutaneous; practical plan for treatment of superficial fungus infections, 313
fungoides, 214, 215, 245, 353, 428
fungoides in tumor stage, 214
fungoides of chest with parapsoriasis en plaques of body, 427
fungoides or acrodermatitis chronica atrophicans with fibrous nodules, 126
fungoides or lymphoblastoma, 234
fungoides; report of case, with clinical, postmortem and experimental observations, *179
Mycosis—Continued
fungoides resembling urticaria pigmentosa, 132
fungoides with bullous lesions; report of case resistant to roentgen and arsenical therapy; effects of empiric therapy, partly based on laboratory investigations, *359
treatment of otomycosis, 660
Myoblastoma, so-called, 348
Myoma, case for diagnosis, 564
Myositis; polymyositis, 422
Myxedema, elephantiasic papulonodular myxedema, *70
localized, 123, 124, 247
Myxoma, effect of temperature on growth and survival of myxoma, herpes and vaccinia viruses in tissue culture, 542
myxomatous degeneration; synovial cysts, 436

Nails, changes in finger nails in pulmonary tuberculosis, *306
dyskeratosis congenita with pigmentation, dystrophia unguis and leukokeratosis oris (Cole and others), 560
dystrophy of, 684
nevus, melanoma or onychomycosis, 134
Polish: See Cosmetics
porokeratosis (Mibelli) associated with cutaneous horn, dystrophy of nails and atrophy of interosseous muscles; report of case, *50
simplified method for staining fungi in skin and in nails, *533
subungual purpura due to neo-synephrin nose drops, 213
Nares: See Nose
National Formulary, plaint, plea and proposal, 200
Necrobiosis Lipoidica Diabeticorum: See Skin, diseases
Necrosis: See under Fat
Negroes, chronic scarring pseudofolliculitis of Negro beard, 312, 539
traumatic marginal alopecia due to traction on hair; comparative study of alopecia liminaris frontalis of Sabouraud, *527
Nelson, N.: Production of bullae in skin of duck; experiments with bullae produced by vesicants, *616
Neoarsphenamine: See Arsphenamines; and under names of various diseases, as Syphilis; etc.
Neocalamine and calamine, 1942, *369
Neoplasms: See Cancer; Sarcoma; Tumors
Neo-Synephrin: See under Nose
Nerves: See Neuritis; Paralysis
Netherton, E. W.: Acrodermatitis chronica atrophicans with fibrous nodules or mycosis fungoides, 126
Acute disseminated lupus erythematosus, 122
Circumscribed scleroderma or lupus erythematosus, 569
Drug-resistant late secondary syphilis, 122
Livedo reticularis, 127
Localized myxedema, 123, 124
Migrating thrombophlebitis following gonorrhea and medication with sulfonamide drugs, 128
Sporotrichosis; localized lymphatic type, 578
Syphilis or papulonecrotic tuberculid, 127
Neuhauser, I.: Foreign body reaction; case for diagnosis, 448
Neuritis, peripheral, lichen planus or dermatitis medicamentosa (gold), 547
Neurodermatitis resembling lymphoblastoma, 437
Neurofibromatosis of palms and wrists; keloids, 246
von Recklinghausen's disease with involvement of scalp, palms and soles; report of case, *656
Neuroses and Psychoneuroses: See also Mental Diseases; etc.
actinomycosis or neurotic excoriations, 550, 562
dermatitis factitia, neurotic excoriations or tuberculids, 218
localized dermatitis herpetiformis or neurotic excoriations, 237
neurotic excoriations, 550
trichotillomania, 227
Nevi, basal cell epithelioma with pigment, secondary to pigmented nevus, 226
cutaneous, with buphthalmos and epilepsy, 664

Nevi—Continued
melanoma, nevus or onychomycosis, 134
moles and malformations of skin, 208
pigmentosus, unusual form of, 228
Vascular: See Angioma
New and Nonofficial Remedies, plaint, plea and proposal, 200
Newborn Infants: See Infants, newborn
Newton, B. L.: Syphilitic juxta-articular nodules, *626
Neymann, C. A.: Alexander Samoilovich Rosenblium; his contribution to fever therapy, *52
Nicholas, L.: Necrobiosis lipoidica diabeticorum with xanthoma cells; report of case, *606
Nicolas-Favre Disease: See Lymphogranuloma Venereum
Nicotinic Acid: See also Vitamins, B
and aphthous stomatitis, 664
Nikolsky's Test: See Pemphigus
Nitroaniline; tetryl dermatitis, 346
Nodes: See also Erythema nodosum; etc.
acrodermatitis chronica atrophicans with fibrous nodules or mycosis fungoides, 126
cutaneous and subcutaneous nodules with juxta-articular infiltrations and rheumatoid symptoms, 425
juxta-articular, of elbows and palms, 130
lupus erythematosus of face associated with nodular infiltrations of back, 577
neurogenic arthropathy of left knee and ankle; gumma of left ankle; juxta-articular nodules due to syphilis; atrophic arthritis, 116
syphilitic juxta-articular nodules, *626
Nomland, R.: Elephantiasis of vulva of undetermined cause; furrowed tongue; perlèche, 221
Treatment of acne vulgaris with comedos by monoterminal electrodesiccation, *302
Norris, C. B.: Favus of glabrous skin and mucous membranes or moniliasis of skin and mucous membranes, 119
Nose, infiltrating basal cell epithelioma, 567
rhinophyma, 570
seborrheic keratosis with epithelioma, 567
subungual purpura due to neo-synephrin nose drops, 213
Nostrums, plaint, plea and proposal, 200

Obermayer, M. E.: Early localized scleroderma: case for diagnosis, 322
Pityriasis lichenoides acuta (Haberman), not varioliform, 321
Post-traumatic degeneration and hyperplasia, 135
Schematic classification of animal organisms causing dermatologic diseases, *512
Obesity, acanthosis nigricans of juvenile type and mild diabetes, 468

OBITUARIES:
Butler, John, 536
Satenstein, David Lawrence, 91
Sellei, Joseph, 537

Occupational Diseases: See Industrial Diseases
Occupations: See Industry and Occupations
O'Farrell, N. M.: Dermatitis venenata due to mistletoe, *416
Oil, Chaulmoogra: See Leprosy
Linseed: See Linseed oil
Mineral: See Petrolatum, liquid
Ointments: See also Rhus, poisoning; Skin, diseases
studies on local action of salicylic acid plus sulfur from various ointment bases, *384
studies on ointments containing resorcinol, *393
studies on penetration of sulfonamides into skin; penetration of sulfonamides from various ointment bases into intact skin of guinea pigs; new method of analysis of tissue sulfonamides, 420
Old Age, effect of estradiol and diethylstilbestrol on atrophic human buccal mucosa, with preliminary report on use of estrogen in management of senile gingivitis, 424
eczema and pruritus in aged, 99
O'Leary, P. A.: Fixed drug eruption produced by diphenylhydantoin sodium, *413
Oleoresin: See Rhus, poisoning
Oliver, E. A.: Amyloidosis of skin, 337
Colloid milium; case for diagnosis, 454
Granuloma inguinale, lymphogranuloma venereum or tuberculosis, 351
Localized dermatitis herpetiformis or neurotic excoriations, 237
Lupus erythematosus of eyelids, 233
Lupus vulgaris with pulmonary tuberculosis, 219
Mycosis fungoides, 215
Necrobiosis lipoidica diabeticorum, 226
Rosacea-like tuberculid of Lewandowsky, 333
Subungual purpura due to neo-synephrin nose drops, 213
Tuberculosis orificialis, 235
Vascular disease; case for diagnosis, 353
Omens, D. V.: Case for diagnosis, 227
Fixed type of toxic erythema, 462
Lichen striatus; case for diagnosis, 449
Livedo reticularis (syphilis), 216
Moniliasis; case for diagnosis, 455
Perifolliculitis capitis abscendens et suffodiens, 226
Psoriasis (resistant); perlèche; scrotal tongue, 460
Recurrent lichen planus, 447
Rosacea-like tuberculid of Lewandowsky; case for diagnosis, 463
Omens, H. D.: Case for diagnosis, 227
Lichen striatus; case for diagnosis, 449
Livedo reticularis (syphilis), 216
Moniliasis; case for diagnosis, 455
Perifolliculitis capitis abscendens et suffodiens, 226
Psoriasis (resistant); perlèche; scrotal tongue, 460
Recurrent lichen planus, 447
Rosacea-like tuberculid of Lewandowsky; case for diagnosis, 463
Omnadin: See Herpes zoster oticus
Onychia: See Nails
Onychodystrophy: See Nails
Onycholysis: See Nails
Onychomycosis: See Nails
Oppenheim, M.: Lupus erythematosus, erythema perstans or erythema elevatum diutinum, 337
Treatment of scabies in wartime, *370
Ormsby, O. S.: Lymphoblastoma, 214
Sarcoidosis, 223
Otsuka, M.: Arthropathic pustular psoriasis, 224
Bowen's disease, 213
Chronic undermining and burrowing ulcer (Meleney), 341
Erythema induratum; case for diagnosis, 212
Lichen planus with recurrent attacks associated with exacerbations of hyperthyroidism, 446
Lupus erythematosus disseminatus subacutus, 452
Mycosis fungoides in tumor stage, 214
Perifolliculitis capitis abscendens et suffodiens, 226
Rosacea-like tuberculid of Lewandowsky; case for diagnosis, 463
Schönlein's purpura or rickettsial infection, 464
Virus diseases of skin with reference to elementary and inclusion bodies; variola-vaccinia and molluscum contagiosum, *635
Oxidase and Peroxidase, peculiar pigmentation following injection of tyrosinase in vitiligo, 110

Paget's Disease: See Vulva
Palate, gumma of upper lip, lower right eyelid and hard palate, 118
Palsy: See Paralysis
Paracoccidioides: See Blastomycosis
Paralysis: See also Extremities, paralysis; etc.
facial; herpes zoster oticus treated with omnadin, 546
Parapsoriasis, 320, 338, 444, 473, 684
mycosis fungoides of chest with parapsoriasis en plaques of body, 427
pityriasis lichenoides acuta (Haberman), not varioliform, 321
Parasites, schematic classification of animal organisms causing dermatologic diseases, *512
Park, R. G.: Hyperhidrosis of feet, 538
Parkes Weber Syndrome: See Hemangiectasis
Patch Test: See Anaphylaxis and Allergy
Peck, S. M.: Vitamin A studies in cases of ichthyosis, *32
Vitamin A studies in cases of keratosis follicularis (Darier's disease), *17

Pediculosis capitis and intelligence in W. A. A. F. recruits, 318
Pemphigus: See also Epidermolysis bullosa
diabetes; pemphigus, 555
dyskeratosis bullosa (so-called benign familial pemphigus), 217
electrolyte content of blister fluid in, 543
erythematosus, 232, 330
foliaceus, 105
of mouth improving with acetarsone therapy, 457
vulgaris, 469
vulgaris-like eruption following use of sulfonamide compounds, 562
vulgaris or dermatitis herpetiformis, 114
Penicillin, clinical use of, 100
Penis, lichen planus; plastic induration of penis (Peyronie's disease), 121
multiple chancriform lesions on, 474
orthostatic lymphedema of penis and scrotum, 670
Pensky, N.: Bubonulus in granuloma inguinale, *494
Exfoliative dermatitis secondary to psoriasis; case for diagnosis, 667
Gummas of leg with syphilis of bones, 242
Halowax acne ("cable rash"); cutaneous eruption in marine electricians due to certain chlorinated naphthalenes and diphenyls, *251
Sarcoid of Boeck with unusual findings; case for diagnosis, 244
Pentamidine: See Leishmaniasis
Periarteritis nodosa, 546
nodosa; urinary sediment in visceral angiitis (periarteritis nodosa, lupus erythematosus, Libman-Sacks "disease"), 422
Perifolliculitis capitis abscendens et suffodiens, 226
Perlèche: See under Lips
Perniosis: See Chilblains
Perspiration: See under Sweat Glands
Petrolatum, liquid; actual causes of certain occupational dermatoses; study of 527 cases with reference to dermatoses due to mineral oils, *579
Petroleum dermatitis; report of 2 cases, *193
Peyronie's Disease: See Penis
Phagocytes and Phagocytosis: See also Reticuloendothelial System
histopathologic study of mode of action of sulfapyridine, 541
Phalanges: See Fingers and Toes
Pharmacopeia of United States, plaint, plea and proposal, 200
Pharynx, syphilitic gumma of, 247
Phenyl Compounds, halowax acne ("cable rash"); cutaneous eruption in marine electricians due to certain chlorinated naphthalenes and diphenyls, *251
Phenytoin, fixed drug eruption produced by diphenylhydantoin sodium, *413
Phialophora: See Fungi
Photosensitization: See under Light
Phthirus Pubis: See Pediculosis
Piedra: See Hair, diseases
Pigmentation: See also Hair; Melanin; Nevi; Schamberg's Disease; etc.
and atrophy of tissues, 666
arsenical, and hyperkeratosis, 547
arsenical, and keratoses, 458
basal cell epithelioma with pigment, secondary to pigmented nevus, 226
case for diagnosis, 227
dyskeratosis congenita with pigmentation, dystrophia unguis and leukokeratosis oris (Cole and others), 560
peculiar pigmentation following injection of tyrosinase in vitiligo, 110
Pink Disease: See Acrodynia
Pinkus, H.: Chronic scarring pseudofolliculitis of Negro beard, 539
Pinta; review of recent etiologic and clinical studies, 420
White: See Vitiligo
Pituitary Body, elephantiasic papulonodular myxedema, *70
Pityriasis, Lichenoides: See Parapsoriasis
rosea, lichen planus or eczema, 439
rubra pilaris, 351
ruba pilaris of familial type; experiences in therapy with carotene and vitamin A, *288
Versicolor: See Tinea versicolor
Plants: See also under names of plants, as Mango; Rhus, poisoning; Tobacco; etc.
Plumbism: See Lead poisoning
Pneumococci, histopathologic study of mode of action of sulfapyridine, 541
Pneumonia, complement fixation for lymphogranuloma venereum and for psittacosis with Frei reactions among pneumonia patients, 541
Poikiloderma atrophicans vasculare (Jacobi), 575
or atrophy of skin, 463
Poison Ivy: See Rhus, poisoning
Poisons and Poisoning: See under names of substances, as Gas; Lead; Mercury; etc.
Polyarteritis Nodosa: See Periarteritis nodosa
Polycythaemia vera accompanied by acne urticata polycythemica and loss of directional power in fingers of left hand, 430
Polymyositis: See Myositis
Popper, H.: Mosaic fungus; cholesterol intercellular artefact, *282
Population, incidence of psoriasis among population at large, *305
Poradenitis: See Lymphogranuloma Venereum
Position: See Posture
Posture, orthostatic lymphedema of penis and scrotum, 670
Powder, phenomenon of "black dermographism," 97
Pregnancy, complications of antisyphilitic therapy in, *83
Protozoa, schematic classification of animal organisms causing dermatologic diseases, *512
Prurigo, studies in abnormal human sensitivity to light; passive transfer of light hypersensitivity in prurigo aestivalis, 543
studies in abnormal human sensitivity to light; photoallergic concept of prurigo aestivalis, 543
studies in abnormal human sensitivity to light; prurigo aestivalis, eczema solare and urticaria photogenica; report of 17 cases and review of literature; light sensitivity in prurigo aestivalis, eczema solare and urticaria photogenica; report of experiments, 206
Pruritus and eczema in aged, 99
blood cholesterol values in cases of generalized pruritis, 663
treatment of essential pruritus and kraurosis vulvae, 424
Pseudofolliculitis, chronic scarring pseudofolliculitis of Negro beard, 312, 539
Pseudomilium, colloid; review of its nomenclature and report of case, *262
Pseudoxanthoma elasticum, 675
elasticum and angioid streaks, *75
Psittacosis, complement fixation for lymphogranuloma venereum and for psittacosis with Frei reactions among pneumonia patients, 541
Psoriasis, *581
arthropathic pustular psoriasis, 224
diabetes; hypertensive cardiovascular disease; psoriasis; scleroderma, 554
erythema multiforme perstans recurrens and psoriasis of palms, 435
exfoliative dermatitis secondary to, 667
gumma of mouth with pseudoepitheliomatous hyperplasia, seborrheic dermatitis or psoriasiform parakeratosis of skin, 574
histopathologic study of, *479
incidence among population at large, *305
lecithin feeding in syndrome of psoriasis; chemical studies of relationship of lecithin feeding to fat metabolism, 544
or lymphoblastoma, 328
pustular; case for diagnosis, 678
resistant; perlèche; scrotal tongue, 460
Psychoneuroses: See Neuroses and Psychoneuroses
Psychoses: See Mental Diseases; Neuroses and Psychoneuroses
Puberty: See Adolescence
Purpura, annular, 229
annularis telangiectodes (Majocchi) and progressive pigmentary dermatosis (Schamberg), 205
Schamberg's progressive pigmentary dermatosis or hypostatic purpura, 329
Schönlein's or rickettsial infection, 464
subungual, due to neo-synephrin nose drops, 213
Pyoderma, *581
gangraenosum; phagedenic ulcer (pyoderma gangraenosum); treatment with sulfapyridine powder and moist chamber therapy, *164
sulfathiazole in treatment in infant, 664
Ulcerosum Tropicalum: See Ulcers

Quackery: See Nostrums

Races: See Negroes; etc.
Radiations: See Roentgen Rays; etc.
Therapy: See Lips, cancer
Radiodermatitis: See under Roentgen Rays
Radium, Therapy: See Hemangioma
Rake, G.: Complement fixation test for lymphogranuloma venereum; results obtained with its use, *619
Rattner, H.: Arsenical pigmentation and keratoses, 458
Lichen planus with atrophy (?), 334
Lupus erythematosus; case for diagnosis, 457
Lupus erythematosus in young child, 330
Lupus erythematosus of eyelid, 339
Recalcitrant eruption of palms, 446
Schamberg's progressive pigmentary dermatosis or hypostatic purpura, 329
Use of urea in hand creams, *47
Raynaud's Disease and generalized progressive scleroderma, 682
Reagins: See Anaphylaxis and Allergy
von Recklinghausen's Disease: See Neurofibromatosis
Recruits: See also Military Medicine
pediculosis capitis and intelligence in W. A. A. F. recruits, 318
Reif, F. B.: Prevention of ringworm of scalp in children, *533
Resin, contact dermatitis due to resin-finished facing in cotton underwear, 95
dermatitis exfoliativa following dermatitis venenata due to shorts, 676, 680
Resorcinol and Resorcinol Derivatives, studies on ointments containing resorcinol, *393
Reticuloendothelial System: See also Phagocytes and Phagocytosis
aleukemic reticuloendotheliosis, 683
Retina, blood supply; angioid streaks and pseudoxanthoma elasticum, *75
Rhinophyma, 570
Rhus, poisoning; evaluation of measures for prevention of ivy dermatitis, *373
poisoning; new base for protective ointment for prevention of poison ivy dermatitis, 313
poisoning; oral prophylaxis against poison ivy; follow-up report, 98
Ribs, cervical, and hyperhidrosis, 317
Rickettsia: See also Rocky Mountain Spotted Fever
morphologic structure of, 424
Schönlein's purpura or rickettsial infection, 464
Ringworm: See also Microsporosis; Mycosis; Tinea; etc.
experimental studies with dermatophytes; influence of age on allergic response in experimental ringworm in guinea pig, 661
inhibitory effect of monochlormercuricarvacrol on growth of pathogenic fungi, *497
microsporon infection of palpebral and ciliary regions, *65
mycoses of feet, 313
prevention of ringworm of scalp in children, *533
Ritter's Disease: See Dermatitis, exfoliative
Robinson, L. B.: Mycosis fungoides; case for diagnosis, 245
Robinson, S. S.: Lupus vulgaris, 137
Rocky Mountain Spotted Fever, morphologic structure of rickettsiae, 424
Roentgen Rays, dermatitis, 244
Therapy: See under names of organs, regions and diseases, as Epithelioma; Lips, cancer; etc.
Roll, H. C.: Multiple idiopathic hemorrhagic sarcoma, 238
Romberger, A. S.: Tetryl dermatitis, 346
Rony, H. R.: Effect of androgen on sebaceous glands of human skin, *601
Rosen, I.: Idiopathic hemorrhagic sarcoma and lymphatic leukemia, 566
Rosenbaum, M. G.: Petroleum dermatitis; report of 2 cases, *193
Rosenblium, Alexander Samoilovich; his contribution to fever therapy, *52
Rossi Test: See Syphilis
Rostenberg, A.: Acanthosis nigricans; case for diagnosis, 109
Rothman, S.: Acanthosis nigricans of juvenile type, mild diabetes and obesity, 468
Annular purpura, 229
Case for diagnosis, 236
Congenital ichthyosiform erythroderma, 458
Kaposi's sarcoma, 466
Rothman, S.—Continued
Pemphigus vulgaris, 469
Solar urticaria, 229
Vitiligo, *400
Rubin, L.: Vitiligo, *400
Rulison, R. H.: Dermatitis in tattooed areas, 557
Sporotrichosis, 556
Russo, J. J.: Dermatitis due to nail polish; report of 2 cases, with experimental studies, *612

Sachs, W.: David Lawrence Satenstein, 91
Pseudoxanthoma elasticum, 675
Sacks-Libman Disease: See Libman-Sacks Disease
Sagher, F.: Phagedenic ulcer (pyoderma gangraenosum); treatment with sulfapyridine powder and moist chamber therapy, *164
Salicyl Compounds, studies on local action of salicylic acid plus sulfur from various ointment bases, *384
Salsberg, R. H.: Dermatitis due to nail polish foundation, 419
Samitz, M. H.: Epidermolysis bullosa; report of case, *159
Sarcoidosis, 223, 552
effect of Schaumann's disease on heart and its mechanism, 546
miliary sarcoid, 223
sarcoid of Boeck with unusual findings, 244
Sarcoma: See also Cancer; Tumors; and under names of tumors
idiopathic hemorrhagic sarcoma and lymphatic leukemia, 566
Kaposi's, 466, 665
Kaposi's, of 15 years' duration, 665
multiple idiopathic hemorrhagic, 135, 238
multiple idiopathic hemorrhagic (Kaposi), 244
Saunders, T. S.: Acute dermatitis due to hair lacquer, 540
Scabies or syphilis, 453
treated with 1 application of benzyl benzoate, 317
treatment by T.E.T.M.S., 318
treatment in wartime, *370
Scalp, cavernous hemangioma treated by interstitial irradiation with low intensity radium needles, 573
Diseases: See under names of diseases, as Ringworm; Seborrhea; Tinea; etc.
myoma; case for diagnosis, 564
pediculosis capitis and intelligence in W.A. A.F. recruits, 318
prevention of ringworm of scalp in children, *533
von Recklinghausen's disease with involvement of scalp, palms and soles; report of case, *656
Scars: See Cicatrix
Schamberg's Disease, purpura annularis telangiectodes (Majocchi) and progressive pigmentary dermatosis (Schamberg), 205
Schamberg's progressive pigmentary dermatosis or hypostatic purpura, 329
Schaumann's Disease: See Sarcoidosis
Scheer, M.: Mycosis fungoides resembling urticaria pigmentosa, 132
Schmidt, F. R.: Colloid degeneration of skin (unilateral), 241
Granuloma annulare, 461
Schönlein Purpura: See under Purpura
Schorr, H. C.: Case for diagnosis, 231
Dermatitis repens, 211
Glomus tumor, 466
Mosaic fungus; cholesterol intercellular artefact, *282
Parapsoriasis; case for diagnosis, 444
Trichotillomania, 227
Scleredema: See Scleroderma
Sclerema: See Scleroderma
Sclerodactylia: See Scleroderma
Scleroderma, circumscribed, or lupus erythematosus, 569
diabetes; hypertensive cardiovascular disease; psoriasis; scleredema, 554
early localized; case for diagnosis, 322
generalized progressive, treated with hyperpyrexia produced by electric blanket method, 568
localized, 568
morphoea guttata, 105
progressive type, 669
Raynaud's disease and generalized progressive scleroderma, 682
Scrofuloderma: See Tuberculosis

Scrotum, orthostatic lymphedema of penis and scrotum, 670
pagetoid epithelioma (scrotum); arsenical keratoses (palms and soles), 319
Scull, R. H.: Chronic recurring lichen planus, 349
Lupus vulgaris, 340
Mycosis fungoides, 214, 353
Sebaceous Glands, adenoma sebaceum treated by solid carbon dioxide, *198
effect of androgen on sebaceous glands of human skin, *601
Seborrhea, cheilitis seborrheica and seborrheic dermatitis of scalp, 136
gumma of mouth with pseudoepitheliomatous hyperplasia, seborrheic dermatitis or psoriasiform parakeratosis of skin, 574
multiple benign cystic epithelioma and seborrheic keratosis, 102
seborrheic dermatitis of eyelids and canities of eyelashes, 324
seborrheic keratosis with epithelioma, 567
Section on Dermatology and Syphilology of American Medical Association, 536
Self-Mutilation, actinomycosis or neurotic excoriations, 550, 562
dermatitis factitia, neurotic excoriations or tuberculids, 218
neurotic excoriations, 550
Selye, H.: Effect of estradiol locally applied to abnormal skin, *188
Semen, studies in transmissibility of syphilis: infectiousness of seminal fluid of men with early syphilis, 544
Senear, F. E.: Case for diagnosis, 236
Dermatitis repens, 211
Foreign body reaction; case for diagnosis, 448
Lupus erythematosus, 222, 331
Lymphoblastoma; case for diagnosis, 335
Multiple idiopathic hemorrhagic sarcoma, 238
Pityriasis rubra pilaris, 351
Senear-Usher Syndrom: Se Pemphigus
Senility: See Old Age
Septum, Nasal: See under Nose
Sesame oil tumors, 97
Sharlit, H.: Erythema multiforme perstans recurrens and psoriasis of palms, 435
Leukoderma treated with intracutaneous injections of tyrosinase enzyme, 430
Sycosis vulgaris or lupus erythematosus, 675
Vitiligo treated with tyrosinase, 438
Shelton, J. M.: Dermatitis due to nail polish foundation, *197
Shipyards: See under Industrial Diseases
Short Waves: See under names of organs and regions
Shorts: See Clothing
Silvers, S. H.: Kaposi's sarcoma of 15 years' duration, 665
Skeer, J.: Atrophy of tissues and pigmentation, 666
Hodgkin's disease with cutaneous manifestations, 106
Skeleton: See Bones
Skin: See also Dermatology
Abnormalities: See also Atrophy; Pigmentation; etc.
abnormalities; moles and malformations of skin, 208
amyloidosis of, 337
apparatus for application of irritant vapors to skin, *173
Atrophy: See Atrophy
bacterial flora of normal skin; study of effect of sulfathiazole and some ointment bases, 544
blood supply; effect of certain vehicles and of salt solutions on reactive hyperemia in skin; application in case of lichen ruber planus, 545
Cancer: See Cancer; Epithelioma; Sarcoma; etc.
colloid degeneration; colloid milium; case for diagnosis, 454
colloid degeneration (unilateral), 241
colloid pseudomilium; review of its nomenclature and report of case, *262
Diseases: See also Dermatitis; and under names of diseases, as Eczema; Herpes; Mycosis; etc.; and under names of plants and drugs, as Arsphenamines; Rhus; etc.
diseases; actual causes of certain occupational dermatoses; study of 527 cases with reference to dermatoses due to mineral oils, *579
diseases; calamine and neocalamine, 1942, *369
Skin—Continued
diseases; course of lectures on occupational dermatoses, 536
diseases; dermatopathic lymphadenitis; focal granulomatous lymphadenitis associated with chronic generalized skin disorders, 206
diseases in war, 317
diseases; necrobiosis lipoidica, 680
diseases; necrobiosis lipoidica diabeticorum, 226
diseases; necrobiosis lipoidica diabeticorum with xanthoma cells; report of case, *606
diseases; photodynamic effects in dermatology, 660
diseases; plaint, plea and proposal, 200
diseases; schematic classification of animal organisms causing dermatologic diseases, *512
diseases; second annual meeting of Consulting Staff of Dermatoses Investigations Section, 202
diseases; sulfonamide therapy in dermatology; observations in 261 cases at Bellevue Hospital, 208
diseases; teaching of occupational dermatoses, *153
disturbances in diabetes mellitus; their relation to vitamin deficiencies, 424
effect of androgen on sebaceous glands of human skin, *601
effect of estradiol locally applied to abnormal skin, *188
Gangrene: See Gangrene
Hemorrhage: See Purpura
Hodgkin's disease with cutaneous manifestations, 106
Hodgkin's disease with specific lesions appearing first in skin, 660
Inflammation: See Dermatitis
manifestations of meningococcic infection, 662
normal and injurious effects of cold on skin and underlying tissues; chilblains and allied conditions, 315
normal and injurious effects of cold on skin and underlying tissues; frostbite, 316
normal and injurious effects of cold on skin and underlying tissues; reactions to cold and injury of normal skin, 315
Pigmentation: See under Pigmentation
production of bullae in skin of duck; experiments with bullae produced by vesicants, *616
production of bullae in skin of duck; preliminary report, *161
relationship between certain physicochemical changes in cornified layer and endogenous bacterial flora of skin, 206
simplified method for staining fungi in skin and in nails, *533
studies on penetration of sulfonamides into skin; penetration of sulfonamides from various ointment bases into intact skin of guinea pigs; new method of analysis of tissue sulfonamides, 420
studies on ointments; local action of salicylic acid plus sulfur from various ointment bases, *384
Tuberculosis: See Tuberculids; Tuberculosis
Tumors: See under Tumors
Ulcers: See Ulcers
variations in cutaneous manifestations of vitamin A deficiency from infancy to puberty, *1
virus diseases of skin with reference to elementary and inclusion bodies; variola-vaccinia and molluscum contagiosum, *635
Skolnik, E. A.: Pemphigus of mouth improving with acetarsone therapy, 457
Slepyan, A. H.: Parkes Weber syndrome; hemangiectatic hypertrophy, 449
So-called myoblastoma, 348
Smallpox, beneficial effect of smallpox vaccine on recurrent aphthous ulcers of mouth and tongue, *151
effect of temperature on growth and survival of myxoma, herpes and vaccinia viruses in tissue culture, 542
virus diseases of skin with reference to elementary and inclusion bodies; variola-vaccinia and molluscum contagiosum, *635
Smith, E. M., Jr.: Blastomycosis, 236
Case for diagnosis, 338
Smith, H. A.: Treatment of scabies in wartime, *370
Smoke, dermatitis venenata caused by, 425
Smoking: See Tobacco

Sobel, N.: Bubonulus in granuloma inguinale, *494
Juxta-articular nodes of elbows and palms, 130
Sobisminol: See Bismuth and Bismuth Compounds
Sobotka, H. H.: Vitamin A studies in cases of keratosis follicularis (Darier's disease), *17
Societies, American Board of Dermatology and Syphilology, 686
Association of Military Surgeons of the United States, 94, 418
Consulting Staff of Dermatoses Investigations Section, second annual meeting, 202
dermatologic, directory of, 687
dermatologic, notice to secretaries of, 94
foreign, directory of, 687
international, directory of, 687
local, directory of, 689
national, directory of, 687
Section on Dermatology and Syphilology of American Medical Association, 536
sectional, directory of, 687
state, directory of, 688

Society Transactions:
Bronx Dermatological Society, 107
Brooklyn Dermatological Society, 105, 242, 665
Chicago Dermatological Society, 210, 328, 444
Cleveland Dermatological Society, 112, 568
Los Angeles Dermatological Society, 134, 319, 470
Manhattan Dermatologic Society, 427, 562, 673
Minnesota Dermatological Society, 102, 681
New York Academy of Medicine, Section of Dermatology and Syphilis, 129, 245, 668
New York Dermatological Society, 547

Sodium chloride: effect of certain vehicles and of salt solutions on reactive hyperemia in skin; application in case of lichen ruber planus, 545
Gold Thiosulfate: See Gold and Gold Compounds
Soil, isolation of Coccidioides from soil and rodents, 313
Solid Carbon Dioxide: See Angioma; Sebaceous Glands
Spencer, G. A.: von Recklinghausen's disease with involvement of scalp, palms and soles; report of case, *656
Spiegel, L.: Localized amyloidosis cutis, 112, 131
Secondary syphilis, 133
Spirochaeta Pallida: See Syphilis
Sporotrichosis, 240, 556, *583
following mosquito bite; description of lesions in girl of Indian and French descent, *525
localized lymphatic type, 578
or tuberculosis, 129
report of cultures of, 341
Spurr, C. L.: Solar urticaria, 229
Stains and Staining, simplified method for staining fungi in skin and in nails, *533
Staphylococci, clinical use of penicillin, 100
Stenhouse, E. E.: Lymphoblastoma (mycosis fungoides?), 234
Stern, E. C.: Lupus erythematosus of face associated with nodular infiltrations of back, 577
Steven, D.: Keratosis follicularis; study of 4 cases, *143
Stevens-Johnson Disease: See Erythema multiforme
Stilbestrol: See Estrogens
Stillians, A. W.: Blastomycosis, 236
Stomatitis, aphthous, and nicotinic acid, 664
aphthous; beneficial effect of smallpox vaccine on recurrent aphthous ulcers of mouth and tongue, *151
Strakosch, E. A.: Studies on ointments; local action of salicylic acid plus sulfur from various ointment bases, *384
Studies on ointments; ointments containing resorcinol, *393
Stratton, E. K.: Mycosis fungoides; report of case, with clinical, postmortem and experimental observations, *179
Streptococci, chronic ulcer of leg caused by microaerophilic hemolytic streptococci (Meleney), 677
microaerophilic hemolytic streptococcus infection of axillas, inguinal and perianal region, 681
Streptococci—Continued
tertiary cutaneous syphilis complicated by secondary infection, possibly by microaerophilic hemolytic streptococci, 132
Stroud, G. M., III: Cavernous hemangioma of scalp treated by interstitial irradiation with low intensity radium needles, 573
Chick embryo antigen (lygranum) test for lymphogranuloma venereum; clinical investigation, *379
Favus of glabrous skin and mucous membranes or moniliasis of skin and mucous membranes, 119
Generalized progressive scleroderma treated with hyperpyrexia produced by electric blanket method, 568
Sudoriparous Glands: See under Sweat Glands
Sulfadiazine: See Sulfonamides; Tularemia
Sulfanilamide: See under Sulfonamides
Sulfonamides, activity of sodium sulfadiazine against Bacterium tularense; effect on growth in vitro, 543
activity of sodium sulfadiazine against Bacterium tularense; treatment of experimental tularemic infection in chick embryo, 543
bacterial flora of normal skin; study of effect of sulfathiazole and some ointment bases, 544
chemotherapeutic prophylaxis with sulfonamide drugs, 420
effect of sulfonamide drugs on trichophytons in vitro, 545
fixed eruption, conjunctivitis and fever from sulfathiazole, *523
histopathologic study of mode of action of sulfapyridine, 541
hypersensitivity to, 98
mechanism of action of certain sulfonamide compounds, 542
migrating thrombophlebitis following gonorrhea and medication with sulfonamide drugs, 128
pemphigus-vulgaris-like eruption following use of sulfonamide compounds, 562
studies on penetration into skin; penetration of sulfonamides from various ointment bases into intact skin of guinea pigs; new method of analysis of tissue sulfonamides, 420
Therapy: See also Dysentery; Gonorrhea; Impetigo; Pneumococci; Pyoderma; Skin, diseases; etc.
therapy in dermatology; observations in 261 cases at Bellevue Hospital, 208
Sulfapyridine: See Pyoderma gangraenosum; Sulfonamides
Sulfathiazole: See Impetigo contagiosa; Pyoderma; Sulfonamides
Sulfur, studies on ointments; local action of salicylic acid plus sulfur from various ointment bases, *384
Sunburn: See Light
Sunlight: See Light
Sunseri, J.: Elephantiasic papulonodular myxedema, *70
Surgery, military; meeting of Association of Military Surgeons, 94, 418
Sutton, R. L., Jr.: Lipoma-like basal cell epithelioma, *176
Sutton's Disease: See Vitiligo
Sweat Glands, cervical rib and hyperhidrosis, 317
experimentally induced disappearance and reappearance of lesions of hydrocystoma, 205
hyperhidrosis of feet, 538
response to acetylcholine, 661
Sweat Shirt: See Clothing
Sweitzer, S. E.: Acrodermatitis chronica atrophicans, 682
Contact dermatitis, 685
Dermatitis herpetiformis, 684
Keratoderma climactericum, 683
Lichen planus, 104
Multiple benign cystic epithelioma, 102
Multiple benign cystic epithelioma and seborrheic keratosis, 102
Parapsoriasis, 684
Ulcerative Hodgkin's disease, 104
Swift-Selter-Feer's Disease: See Acrodynia
Sycosis vulgaris or lupus erythematosus, 675
Synovial Membrane, myxomatous degeneration; synovial cysts, 436
Syphilis: See also Chancre; Venereal Diseases; and under names of organs, regions and diseases, as Aortitis; Bones; Eyes; etc.
blood level and excretion of arsenic following single injections of clorarsen, *297

Syphilis—Continued
case for diagnosis subsequently proved to be 1 of syphilis, 453, 469
cerebral reactions associated with massive mapharsen treatment of early syphilis, 204
complications of antisyphilitic therapy in pregnancy, *83
drug-resistant late secondary syphilis, 122
early; urticaria pigmentosa, 460
false positive serologic tests in children, 208
field laboratory test of Briceño Rossi; new reaction for diagnosis of syphilis, 209
further evidence concerning specificity of lymphogranuloma venereum complement-fixation test in syphilis, 421
granuloma inguinale; late latent syphilis, 112
importance of time factor in evaluation of "cure" in syphilitic rabbits, 204
intravenous administration of sobisminol solution; toxicity in normal and in syphilitic rabbits, *35
livedo reticularis (syphilis), 216
neurogenic arthropathy of left knee and ankle; gumma of left ankle; juxta-articular nodules due to syphilis; atrophic arthritis, 116
or papulonecrotic tuberculid, 127
or scabies, 453
primary; preliminary intensive treatment by daily injection of arsenicals, 204
pulmonary fat embolism following infusions via bone marrow, 205
reinfection in, 421
removal of divalent cations from solution by beef heart antigens, 206
resistant (nonresistant in wife), *412
secondary, 133
sensitivity and specificity of Kahn reaction in temperature range from 37° C. through 56° C with syphilitic sera with consideration of value of Kahn verification test in syphilis, 204
serologic (nonsyphilitic) reaction approaching universal sensitivity, 662
studies in transmissibility; infectiousness of seminal fluid of men with early syphilis, 544
superficial nonulcerative; case for diagnosis, 452
syphilitic gumma of eye, 672
syphilitic gumma of pharynx, 247
syphilitic juxta-articular nodules, *626
tertiary; arthropathy of hands and wrists with multiple areas of cystic degeneration of bone, 117
tertiary cutaneous syphilis complicated by secondary infection, possibly by microaerophilic hemolytic streptococci, 132
Syphiloma: See Syphilis, tertiary

T. E. T. M. S.: See under Scabies
Tabes Dorsalis, intravenous thiamine chloride (vitamin B_1) in treatment of tabetic lightning pains, 204
Tattooing, dermatitis in tattooed areas, 557
Taylor, H. C.: Cutaneous blastomycosis complicated by meningitis, *88
Teaching: See Education
Tear Gas: See Gas, poisoning
Teeth, prosthetic ulcer of mouth, 314
Temperature: See also Cold
effect on growth and survival of myxoma, herpes and vaccinia viruses in tissue culture, 542
Templeton, H. J.: Resistant syphilis (nonresistant in wife), *412
Testosterone: See Androgens
Tetryl: See Nitroaniline
Thiamine: See also Vitamins, B
intravenous thiamine chloride (vitamin B_1) in treatment of tabetic lightning pains, 204
Thigh, ulcers; ulcerative Hodgkin's disease, 104
Thorax: See also Heart; Ribs; etc.
mycosis fungoides of chest with parapsoriasis en plaques of body, 427
Thrombocytopenia: See Purpura
Thrombophlebitis, migrating, following gonorrhea and medication with sulfonamide drugs, 128
Thrush: See Moniliasis
Thyroid, hyperthyroidism, lichen planus with recurrent attacks associated with exacerbations of, 446
Ticks: See Rocky Mountain Spotted Fever
Tinea: See also Ringworm
treatment with ethyl chloride, *511
versicolor, lichen planus or moniliasis, 561
Tissue: See also Cells
Adipose: See Fat
studies on penetration of sulfonamides into skin; penetration of sulfonamides from various ointment bases into intact skin of guinea pigs; new method of analysis of tissue sulfonamides, 420
Tobacco skin-reactions in peripheral vascular diseases and coronary artery disease, 206
Toe Nails: See Nails
Toes: See Fingers and Toes
Tongue, beneficial effect of smallpox vaccine on recurrent aphthous ulcers of mouth and tongue, *151
black hairy tongue, 432
elephantiasis of vulva of undetermined cause; furrowed tongue; perlèche, 221
psoriasis (resistant); perlèche; scrotal tongue, 460
Toxicodermas: See Dermatitis, venenata; Drugs, eruptions
Tragacanth, allergenic properties of vegetable gums; case of asthma due to tragacanth, 423
Traub, E. F.: Angiomas of head, 561
Paget's disease of vulva, 559
Pemphigus; diabetes, 555
Sickle cell anemia with ulcerations, 558
Tuberculid of face (rosacea-like), 555
Xanthoma tuberosum multiplex, 553
Trauma, herpes zoster traumaticus, *581
post-traumatic degeneration and hyperplasia, 135
Treponema Pallidum: See Syphilis
Trichophyton, effect of sulfonamide drugs on trichophytons in vitro, 545
Trichorrhexis Nodosa: See Hair, diseases
Trichotillomania, 227
Trinitrophenylmethylnitramine: See Nitroaniline
Tropical Medicine: See also under names of tropical diseases, as Leishmaniasis; Ulcers, tropical; etc.
Tubercle Bacilli: See also Tuberculosis
death of tubercle bacilli subjected to oxygen deprivation in presence of moisture and of warmth, 542
Tuberculids: See also under Tuberculosis
dermatitis factitia or neurotic excoriations, 218
papulonecrotic, or syphilis, 127
rosacea-like, of face, 555
rosacea-like, of Lewandowsky, 333, 463
Tuberculin: See under Tuberculosis
Patch Test: See Tuberculosis
Therapy: See Lupus erythematosus tumidus superficialis
Tuberculosis: See also Tuberculids; and under names of diseases, organs and regions, as Mouth; etc.
asphyxiated bacteria as vaccine in, 542
cutis; some types observed in Buenos Aires, 209
granuloma inguinale, lymphogranuloma venereum or tuberculosis, 351
histopathology of Mantoux reaction and erythema nodosum, 318
of cutis and hypoderm associated with genitourinary tuberculosis and chronic pulmonary tuberculosis, 249
of skin, 218
or sporotrichosis, 129
orificialis, 235
pulmonary, and lupus erythematosus, 103
pulmonary, changes in finger nails in, *306
pulmonary; erythema induratum and advanced pulmonary tuberculosis, 103
pulmonary; necrobiosis lipoidica diabeticorum with xanthoma cells; report of case, *606
pulmonary; tuberculosis of cutis and hypoderm associated with genitourinary tuberculosis and chronic pulmonary tuberculosis, 249
pulmonary, with lupus vulgaris, 219
verrucosa cutis; case for diagnosis, 231
Tularemia, activity of sodium sulfadiazine against Bacterium tularense; effect on growth in vitro, 543
activity of sodium sulfadiazine against Bacterium tularense; treatment of experimental tularemic infection in chick embryo, 543
Tulipan, L.: Localized myxedema, 247
Tumors: See also Adenoma; Angioma; Cancer; Epithelioma; Fibroma; Hemangioma; Lipoma; Lymphangioma; Lymphoblastoma; Melanoma; Myxoma; Rhinophyma; Sarcoma; etc.; and under names of organs and regions as Arms; Nose; etc.
glomus tumor, 466

Tumors—Continued
lupus erythematosus timidus superficialis; treatment with tuberculin; report of case, *60
mycosis fungoides in tumor stage, 214
Tyroglyphus Longior: See Acariasis
Tyrosinase: See Enzymes; Oxidase and Peroxidase

Ulcers: See also under names of organs and regions, as Mouth; Thigh; etc.
chronic, of leg caused by microaerophilic hemolytic streptococci (Meleney), 677
chronic undermining and burrowing ulcer (Meleney), 341
multiple extensive ulcerations of legs, 327
multiple, of skin; chronic ulcerative colitis, 551
of legs, *581
sickle cell anemia with ulcerations, 558
treatment of leg ulcers with blood and concentrated plasma, 420
tropical; pyoderma ulcerosum tropicalum, 425
tropical, severe, *414
Ultrashort Waves: See under names of organs, regions and diseases
Ultraviolet Rays: See Light
Therapy: See under names of organs, regions and diseases
Umansky, M.: Erythema palmare (red palms), 107
Underwear: See Clothing
Urea, use in hand creams, *47
Urine, urinary sediment in visceral angiitis (periarteritis nodosa, lupus erythematosus, Libman-Sacks "disease"), 422
Urticaria, emotional settings of some attacks, of, 100
papulosa; lichen urticatus, 102
pigmentosa, 102
pigmentosa; early syphilis (?), 460
pigmentosa in adult, 558
pigmentosa resembling mycosis fungoides, 132
solar, 229
studies in abnormal human sensitivity to light; prurigo aestivalis, eczema solare and urticaria photogenica; report of 17 cases and review of literature; light sensitivity in prurigo aestivalis, eczema solare and urticaria photogenica; report of experiments, 206
Usher-Senear Syndrome: See Pemphigus

Vaccinia: See Smallpox
Van Dyck, L. S.: Dermatitis due to nail polish: report of 2 cases, with experimental studies, *612
Vapors, apparatus for application of irritant vapors to skin, *173
Variola: See Smallpox
Venereal Diseases: See also Chancre; Gonorrhea; Syphilis; etc.
control in army, 101
Vero, F.: Lichen planus et atrophicus (folliculitis decalvans and lichen spinulosus, Little and McCafferty), 669
Nevus, melanoma or onychomycosis, 134
Verruca, Tuberculosis: See Tuberculosis
Vesication, acquired hypersensitivity to chlorinated ethylamine vesicants; report of case, *15
mycosis fungoides with bullous lesions; report of case resistant to roentgen and arsenical therapy; effects of empiric therapy, partly based on laboratory investigations, *359
production of bullae in skin of duck; experiments with bullae produced by vesicants, *616
production of bullae in skin of duck; preliminary report, *161
Viruses: See also under Herpes; Lymphogranuloma Venereum; Myxoma; Rabies; etc.
diseases of skin with reference to elementary and inclusion bodies; variola-vaccinia and molluscum contagiosum, *635
Vision, chemotherapeutic prophylaxis with sulfonamide drugs, 420
Vitamins: See also Nicotinic Acid; Thiamine; etc.
A: See also Carotene
A deficiency; variations in cutaneous manifestations from infancy to puberty, *1
A: effects of depletion in young adults, 422
A: keratosis follicularis; study of 4 cases, *143
Vitamins—Continued
A; pityriasis rubra pilaris of familial type; experiences in therapy with carotene and vitamin A, *288
A studies in cases of ichthyosis, *32
A studies in cases of keratosis follicularis (Darier's disease), *17
acanthosis nigricans; is it form of avitaminosis? *650
B: See also Nicotinic Acid; Thiamine
B; biotin and skin sensitivity, 99
B_1: See Thiamine
skin disturbances in diabetes mellitus; their relation to vitamin deficiencies, 424
Vitiligo, *400
in 3 siblings, *519
leukoderma treated with intracutaneous injections of tyrosinase enzyme, 430
of upper lip in players of wind instruments, *605
peculiar pigmentation following injection of tyrosinase in, 110
treated with tyrosinase, 438
Vulva, elephantiasis of undetermined cause; furrowed tongue; perlèche, 221
Paget's disease of, 559
treatment of essential pruritus and kraurosis vulvae, 424

Walzer, A.: Pemphigus foliaceus, 105
War: See also Military Medicine; etc.
dermatologists in, 417
Wassermann Reaction: See Syphilis
Weber, L. F.: Contact dermatitis due to sweat shirt, 239
Weber's Disease: See Hemangiectasis
Weichselbaum, P. K.: Lupus erythematosus, 222
Weiner, A. L.: Pityriasis rubra pilaris of familial type; experiences in therapy with carotene and vitamin A, *288
Werner Syndrome: See Scleroderma
Wheal: See Urticaria
White Spot Disease: See Scleroderma
Wilhelm, L. F. X.: Arsenical dermatitis with superficial epitheliomatosis, 324
Wise, F.: Dyskeratosis congenita with pigmentation, dystrophia unguis and leukokeratosis oris (Cole and others), 560
Extensive postacne keloids, 431
Lepra; case for diagnosis, 557
Multiple ulcers of skin; chronic ulcerative colitis, 551
Mycosis fungoides with bullous lesions; report of case resistant to roentgen and arsenical therapy; effects of empiric therapy, partly based on laboratory investigations, *359
Urticaria pigmentosa in adult, 558
Wolf, J.: Basal cell epithelioma, 437
Basal cell epithelioma of left axilla and left arm, 677
Lichen planus, pityriasis rosea or eczema, 439
Lichen sclerosus et atrophicus, 430, 433
Myoma; case for diagnosis, 564
Workmen's Compensation and Insurance: See also Industrial Diseases
actual causes of certain occupational dermatoses; study of 527 cases with reference to dermatoses due to mineral oils, *579
Wrist, neurofibromatosis of palms and wrists; keloids, 246

Xanthoma, case for diagnosis, 471
multiple xanthomatosis tuberosa, 664
necrobiosis lipoidica diabeticorum with xanthoma cells; report of case, *606
tuberosum multiplex, 553
Xanthomatosis: See Xanthoma
X-Rays: See Roentgen Rays

Yeast: See Ringworm

Zakon, S. J.: Alexander Samoilovich Rosenblium; his contribution to fever therapy, *52
Effect of androgen on sebaceous glands of human skin, *601
Pemphigus erythematosus, 232
Pemphigus of mouth improving with acetarsone therapy, 457
Rosacea-like tuberculid of Lewandowsky, 333
Subungual purpura due to neo-synephrin nose drops, 213
Zona: See Herpes zoster
Zoster: See Herpes zoster

JAN 1 1944

Hospital Library

VOLUME 48 NUMBER 6

ARCHIVES
OF
DERMATOLOGY AND SYPHILOLOGY

EDITORIAL BOARD

HOWARD FOX, Chief Editor
[illegible] East Fifty-Fourth Street, New York

C. C. DENNIE, Kansas City, Mo.
HAROLD N. COLE, Cleveland
HERBERT RATTNER, Chicago
FRED D. WEIDMAN, Philadelphia
GEORGE M. MacKEE, New York
C. GUY LANE, Boston

DECEMBER 1943

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second Class Matter Oct. 30, 1917, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

COPYRIGHT, 1943, BY THE AMERICAN MEDICAL ASSOCIATION

THE ARCHIVES OF [illegible] AND SYPHILOLOGY is published by the American Medical Association to [illegible] and to disseminate knowledge in these departments of medicine.

Manuscripts for publication, for review and correspondence relating to the editorial management should be addressed to Howard Fox, Chief Editor, 140 East Fifty-Fourth Street, New York, or to any member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed: ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY, 535 North Dearborn Street, Chicago 10.

Articles are accepted for publication on condition that they are contributed solely to the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY. Manuscripts must be typewritten, preferably double spaced, and [illegible] should be submitted. Zinc etchings and halftones will be supplied by the Association when the original illustrations warrant reproduction and when their number is not excessive.

Footnotes and bibliographies (the latter are used only in exhaustive reviews of the literature) should conform to the style of the *Quarterly Cumulative Index Medicus*. This requires, in the order given: [illegible], title of article and name of periodical, with volume, page, month—day of month if journal appears weekly—and year.

Matter appearing in the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY is covered by copyright, but as a rule no objection will be made to its reproduction in a reputable medical journal if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY or in any of the other publications issued by the Association will not be permitted.

The ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY is published monthly. The annual subscription price (for two volumes) is as follows: domestic, $8.00; Canadian, $8.40; foreign, $9.00, including postage. Single copies are 85 cents, postpaid.

Checks, money orders, etc., should be made payable to the American Medical Association.

OTHER PERIODICAL PUBLICATIONS
of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general interest to the profession. Illustrated. Annual subscription price (three volumes): domestic, $8.00; Canadian, $8.80; foreign, $9.00. Single copies, 25 cents.

AMERICAN JOURNAL OF DISEASES OF CHILDREN—Monthly. Presents pediatrics as a medical science and as a social problem. Original articles, specially prepared reviews, based on recent pediatric literature, abstracts from foreign and domestic journals, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 85 cents.

ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced original clinical and laboratory investigations in the field of internal medicine. Illustrated. Annual subscription price (two volumes): domestic, $5.00; Canadian, $5.40; foreign, $6.00. Single copies, 75 cents.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 85 cents.

ARCHIVES OF SURGERY—Monthly. Devoted largely to the investigative and clinical phases of surgery, with monthly reviews of progress in urologic surgery. Well illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 85 cents, except special numbers.

ARCHIVES OF OPHTHALMOLOGY—Monthly. A medium for the presentation of original articles on diseases of the eye, with abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents.

ARCHIVES OF OTOLARYNGOLOGY—Monthly. A periodical devoted to the publication of original articles and general reviews in the field of otolaryngology. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents, except special issues.

ARCHIVES OF OPHTHALMOLOGY—Monthly. Includes original articles on diseases of the eye, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $9.00. Single copies, 85 cents.

WAR MEDICINE—Monthly. Devoted to materials on progress and scientific advancement in relation to military, naval, public health and allied services. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the world's current medical literature. Issued four times a year, each cumulating the fourth volume bound for permanent reference. Annual subscription price calendar year: domestic, $12.00; Canadian, $14.00; foreign, $14.00.

AMERICAN MEDICAL ASSOCIATION
535 North Dearborn Street CHICAGO 10

CONTENTS

PAGE

Actual Causes of Certain Occupational Dermatoses: A Study of 527 Cases, with Special Reference to Dermatoses Due to Mineral Oils. Joseph V. Klauder, M.D., Philadelphia 579

Effect of Androgen on the Sebaceous Glands of Human Skin. Hugo R. Rony, M.D., and Samuel J. Zakon, M.D., Chicago 601

Vitiligo of Upper Lip in Players of Wind Instruments. C. Wimberly Freeman, M.D., and H. H. Hazen, M.D., Washington, D. C. 605

Necrobiosis Lipoidica Diabeticorum with Xanthoma Cells: Report of a Case. Leslie Nicholas, M.D., Philadelphia 606

Dermatitis Due to Nail Polish: Report of Two Cases, with Experimental Studies. Harry Keil, M.D., New York; Joseph J. Rusko, M.D., Albany, N. Y.; and Laird S. Van Dyck, M.D., New York 612

Production of Bullae in the Skin of the Duck: II. Experiments with Bullae Produced by Vesicants. Leon Goldman, M.D.; Norton Nelson, Ph.D., and I. Arthur Mirsky, M.D., Cincinnati 616

Complement Fixation Test for Lymphogranuloma Venereum: Results Obtained with Its Use. Arthur W. Grace, M.D., Brooklyn, and Geoffrey Rake, M.B., B.S., New Brunswick, N. J. 619

Syphilitic Juxta-Articular Nodules. Frederick Kalz, M.D., and B. L. Newton, M.D., Montreal, Canada 626

Virus Diseases of Skin, with Special Reference to Elementary and Inclusion Bodies: I. Variola-Vaccinia and Molluscum Contagiosum. Michael H. Ebert, M.D., and M. Otsuka, M.D., Chicago 635

Acanthosis Nigricans: Is It a Form of Avitaminosis? Lester Hollander, M.D., Pittsburgh 650

Clinical Notes:

Allergic Dermatitis Caused by Hair Lacquer Products. Alfred Hollander, M.D., Springfield, Mass. 656

Von Recklinghausen's Disease with Involvement of the Scalp, Palms and Soles: Report of a Case. Gerald A. Spencer, M.D., New York 656

Correspondence:

The Chick Embryo Antigen (Lychnanum) Test for Lymphogranuloma Venereum. Arthur W. Grace, M.D., Brooklyn 659

Abstracts from Current Literature 660

Society Transactions:

Brooklyn Dermatological Society 665

New York Academy of Medicine, Section of Dermatology and Syphilis 668

Manhattan Dermatologic Society 673

Minnesota Dermatological Society 681

News and Comment 686

Book Reviews 686

Directory of Dermatologic Societies 687

General Index 691